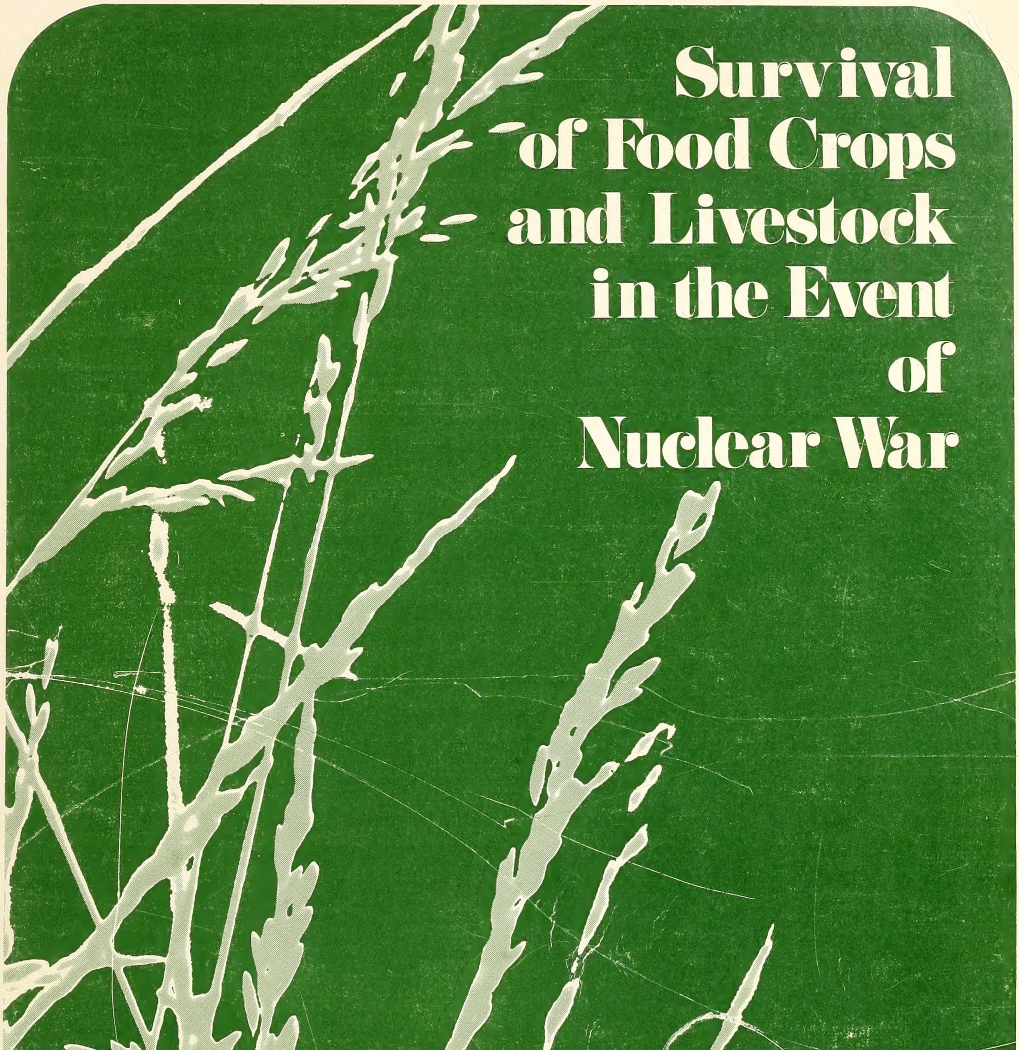
AEC Symposium Series 24



Survival of Food Crops and Livestock in the Event of Nuclear War

U. S. ATOMIC ENERGY COMMISSION Office of Information Services

BETA-RADIATION DOSES FROM FALLOUT PARTICLES DEPOSITED ON THE SKIN

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ABSTRACT

Absorbed beta-radiation dose expected from fallout particles deposited on the skin was estimated by use of the Beta Transmission, Degradation, and Dissipation (TDD) model. Comparison of computed doses with the most recent experimental data relative to skin response to beta-energy deposition leads to the conclusion that, even for fallout arrival times as early as 10^3 sec (16.7 min postdetonation), no skin ulceration is expected from single particles 500 μ or less in diameter.

Doses from arrays of fallout particles of different size distributions were computed also for several fallout-mass deposition densities; time intervals required to accumulate doses sufficient to initiate skin lesions were calculated.

In 1954 residents of Rongelap Atoll in the Marshall Islands were exposed to fallout arriving within hours after detonation of the Castle Bravo nuclear device. Several of the atoll's inhabitants suffered severe skin burns. Primarily as a result of this experience, the possibility of "beta burn" from nuclear fallout has been recognized. However, to date, attempts to predict the acute or chronic skin effects that might be expected following exposure to fallout have been limited. This limitation results mainly from the lack of experimental data on the biologic response of the skin to particulate-source exposures, from incomplete understanding of the relation of such response to that encountered in other localized exposures (e.g., collimated X-ray beams) for which data are available, and from the absence of reliable beta-dose calculational models. All these are required to relate dose to observed effect in a manner allowing prediction of the biological effects from knowledge of the expected fallout interaction.

The literature indicates that work on the theoretical aspects of the beta-dose problem has progressed faster than have experimental efforts. As early as 1956 Loevinger, Japha, and Brownell devised an analytical representation (model) to calculate beta doses from "discrete radioisotope sources."[1] By 1966 four models were available.[2] The most precise, though complex, of these models is the Transmission, Degradation, and Dissipation (TDD) model.[2] This paper is based on the TDD model and presents predicted beta doses that would result from skin deposition of nuclear-weapon fallout particles.

A nuclear attack on the United States would be expected to result in low-intensity gamma-radiation fields over much of the fallout area that would develop. Exposure to the low-intensity field would pose little or no immediate or long-term whole-body gamma-radiation hazard. However, it has been suggested that in such situations contact of individual fallout particles with exposed skin could constitute a potential hazard. Individual particles can deposit on the skin via direct deposition during passage of the fallout cloud or following resuspension of particles at a later time.

Each particle, if radioactive enough, is capable of producing a lesion. If several particles reside close enough in the same general skin area, their effects could be additive, in the sense of causing one lesion. However, at larger particle-separation distances, beta-radiation dose deliveries would not interact. That is, the dose contribution from one particle to the tissue in the vicinity of another particle would be negligible. This situation is treated separately in the next three sections. At small particle-separation distances, estimation of the dose delivered at any point in tissue would require summation of the dose contributions from all particles in the immediate vicinity. This latter situation is treated separately also.

THE SINGLE-PARTICLE BETA-DOSE MODEL

The TDD model for single particles is composed of six separate semi-independent computer codes. The first (Code 1) is a nuclide-abundance code that calculates the activity of each radionuclide generated in the detonation of a nuclear device or weapon. This code also considers radioactive decay and calculates fission-nuclide activity at any postdetonation time.

Code 2 computes the beta spectrum for each beta-emitting nuclide, given the end-point energies, beta branching fractions, and degree of forbiddenness of the beta transitions.[3] Output from this code is a sequence of values representing the probability that a beta particle will be emitted with an energy between E and $E + \Delta E$, where $\Delta E = 0.04$ MeV and values for E range from 0 to the maximum β energy, E_{max}. Individual fission-product beta spectra have been generated and are stored on tape for use with the composite-spectrum code (Code 3).

Code 3 is a composite-spectrum routine that sums the individual beta spectra of the fission-product nuclides with appropriate weighting for the activity of each contributing nuclide, as determined by Code 1. Code 3 produces a point-source beta spectrum at a given time for the specific weapon under consideration. Output from this code is a sequence of values representing the number of betas per energy interval emitted by the source.

The electron spectrum from a fallout particle (assumed to be spherical in shape) differs from that produced by a point source because scattering and absorption processes within the particle degrade the spectrum. Calculation of the extent of degradation is complicated by the fact that in fallout particles some fission products are uniformly distributed within the particle material, others have condensed on the particle surface, and the rest behave in an intermediate fashion.[4]

Korts and Norman developed a model,[5] termed the Condensed State Diffusion Controlled Model, which describes the mechanism of fission-product absorption in fallout material distributed in a radioactive cloud following a nuclear detonation. In this model they assumed that (1) the fallout material is glassy silicate; (2) the surface of a fallout particle is in equilibrium with the gas phase; and (3) the rate of transfer of fission products into the interior of the fallout particle is diffusion controlled. One output of this Condensed State Diffusion Controlled Model consists of a set of radial fission-product-concentration profiles in fallout particles of different sizes. Using such concentration profiles, Korts and Norman calculated for each fission product the percentage of total nuclide present which would diffuse into the particle. In almost all cases examined, they found that "loadings" of 0, 25, 50, 62.5, 75, 82.5, and 100% (by weight) could be used to describe the portion of fission product present diffusing into the particle. (The complementary percentage in each of the seven classes represents the portion of the fission product present that remains at the particle surface.) Zero percent diffusion takes place when the fission product condenses on the particle surface, essentially without any diffusion during particle cooling; whereas 100% diffusion represents complete diffusion leading to homogeneous distribution of the fission product in the silicate matrix. This Condensed State Diffusion Controlled Model was used in the manner described in the following paragraph to provide the geometric basis for the electron degradation within fallout particles.

Degradation suffered by the emanating electron spectrum is handled by Code 4, a Monte Carlo program that starts with a given number of emitted betas in a specified energy interval and then computes the loss in electron energy and number due to scattering and absorption processes within the particle. The code outputs two sets of Monte Carlo determined energy-dependent loss coefficients, set A for homogeneously distributed fission products and set B for surface-condensed fission products. These coefficients are then applied to the composite beta spectrum from the point source of fission products (Code 3) by Code 5. Application of these loss factors is straightforward for the 0 and 100% diffusion cases (in which set B and set A, respectively, are utilized). For five intermediate diffusion cases, set A was applied to the percentage diffusing into the particle, and set B was applied to the percentage remaining at the surface. Output of Code 5 thus consists of a degraded beta spectrum emerging from a fallout particle of a specified size.

Code 6 operates on the resulting composite degraded spectrum to compute the depth-dose rate in tissue. This is based on energy-dissipation factors for fast electrons as calculated by Spencer.[6]

The dose rate, D_t (in rads per hour), at a tissue depth Z centimeters from a particle of volume V (in cubic centimeters) emitting $N_e(E_0)$ beta particles per second per cubic centimeter in the energy interval ΔE with mean energy E_0 (in million electron volts) (this is the emerging degraded spectrum in the present work) is given by:

$$D_t = \frac{kfgV}{4\pi Y^2} \sum_{E_0=\Delta E/2}^{E_0=E_{max}-\Delta E/2} J(x)\,(dE/dr)_{E_0}\, N_e(E_0) \qquad (1)$$

where k = a constant, 5.76×10^{-5} (rad-g-sec)/(MeV-hr), relating energy-transport rate to dose rate

f = dimensionless correction factor for a semi-infinite absorber, determined from an auxiliary Monte Carlo program

g = ratio of dose rate at a distance Y (in centimeters) from the center of a spherical source (radius R in centimeters) to the dose rate from a point source at the same distance ($Y > R$); the ratio is a dimensionless quantity given by

$$g = \frac{3Y^2}{R^2}\left[0.5 + \left(\frac{R^2 - Y^2}{4RY}\right) \ln \left(\frac{Y + R}{Y - R}\right)\right] \qquad (2)$$

J(x) = Spencer's energy-dissipation-distribution function evaluated at tissue depth Z measured in units of the normalizing residual range, r_0; $x = z\rho/r_0$, ρ being the density of the absorbing medium[6]

$(dE/dr)_{E_0}$ = stopping power of the absorber for electrons emitted from the particle with energy E_0

The resulting dose rates, summed over the composite degraded spectrum, form the output of this part of the model.

The final operation of the composite TDD model integrates the various dose rates (from each energy interval) computed via Eq. 1 over time to get the total absorbed dose. In practice, to reduce computation time, we carry out the integration by the use of time-integrated beta activities derived from the inventory code (Code 1) to make up a time-integrated composite beta spectrum. This spectrum is then degraded and deposited in tissue as explained previously; i.e., the time integration is done from the start rather than as the last step.

Recently the six codes have been unified into a single modified composite program to reduce computer run time.[7] Also, several new features have been introduced into the composite program to increase its ability to cope with a variety of beta-dose problems.[8]

EVALUATION OF THE SINGLE-PARTICLE MODEL

Validity of the TDD-model dose predictions has been examined[8] by comparing the model-computed doses delivered by reactor-irradiated UC_2 particles with (1) doses from the UC_2 particles measured with a β-extrapolation chamber;[9] (2) values for UC_2 particle dose obtained by a photographic-film dosimetry technique; and (3) dose values computed by applying a completely independent Monte Carlo calculational technique.

Tests included doses at shallow as well as at relatively large depths (7500 μ) in tissue and at points directly underneath the particle and points radially displaced to distances as far as 5000 μ. Particles of variable sizes and reactor irradiation times of different duration were also included in the comparisons.

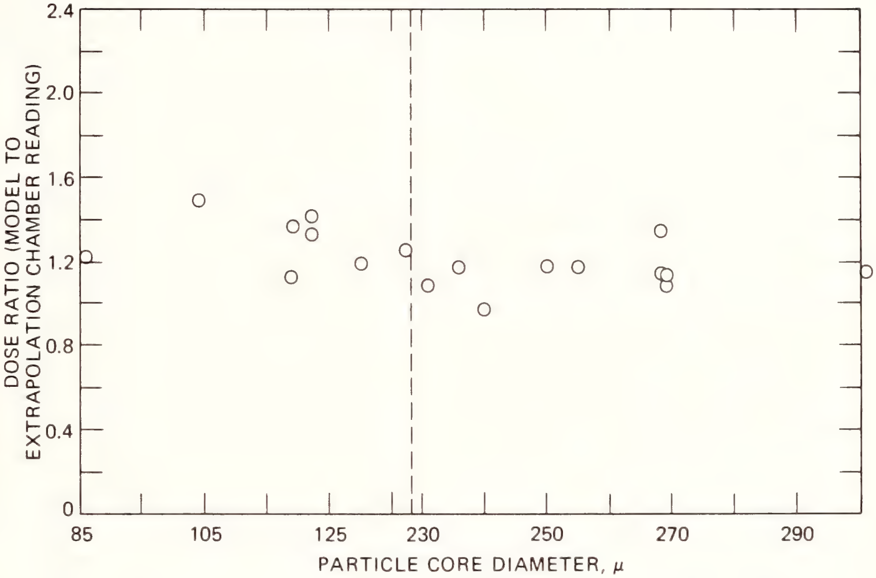

Fig. 1 Ratio of calculated (TDD model) dose to dose measured with a β-extrapolation chamber (tissue depth, 30 μ).

Typical results obtained in the comparisons with data from the extrapolation chamber, the Monte Carlo program, and the photographic-film exposure technique are presented in Figs. 1, 2, and 3, respectively. The primary conclusions drawn from the comparisons were:[8]

1. On the whole, agreement between values obtained by use of the composite program and those obtained by experimentation and exercise of the cited Monte Carlo program was satisfactory.

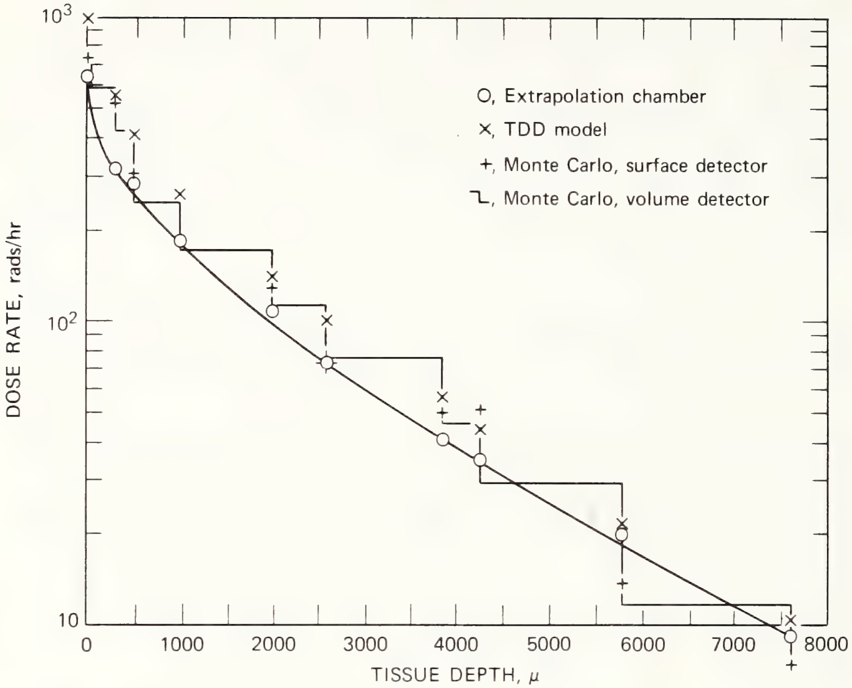

Fig. 2 Comparison of TDD model calculations with Monte Carlo calculations and extrapolation-chamber measurements (delay time, 5.75 hr).

2. The ranges of particle sizes (85 to 310 μ) and time periods of reactor irradiation (5 min to 24 hr) considered appear to have little influence on the extent of agreement achieved.

3. Tissue beta-radiation delivery (i.e., absorption) estimated by the composite TDD model for shallow tissue depths is invariably higher than that derived from the Monte Carlo calculations. As the tissue depth considered increases, agreement between the TDD model and experimental results improves until, as shown in Fig. 4, at a tissue path length of about 4000 μ the values for the model and those for the test method tend to agree. Such relations are interpreted to indicate that the model underestimates electron attenuation in the particle material and overestimates that in tissue.

4. Delay times (time periods between termination of reactor irradiation and start of tissue exposure) greater than approximately 25 hr appear to increase the difference between model predictions and values determined by the test methods, but not to an appreciable degree.

5. Doses measured directly below the particle by photographic-film experiments agree rather well with model predictions, except for dose locations very close to the particle, in which case apparent saturation of film occurs.

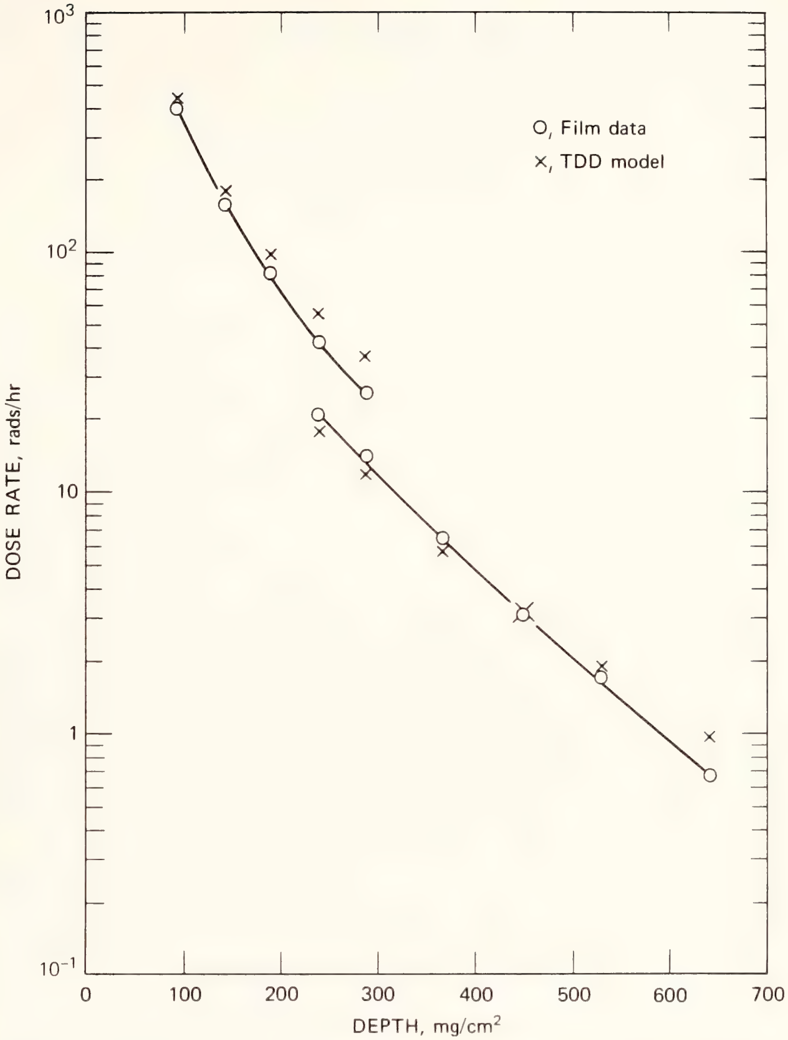

Fig. 3 Comparison of calculated (TDD model) dose rates with film data (160-μ particles).

DOSE CRITERIA FOR SINGLE-PARTICLE EXPOSURE

Serious acute lesions of the skin are induced primarily by the destruction of the germinal cells of the epithelium. In humans the subsurface depth of the skin germinal-cell layer varies from 20 to 250 μ. However, for convenience a single depth of 100 μ is usually chosen to represent the critical level. The absorbed beta dose (or amount of beta energy absorbed in an infinitesimally small mass of

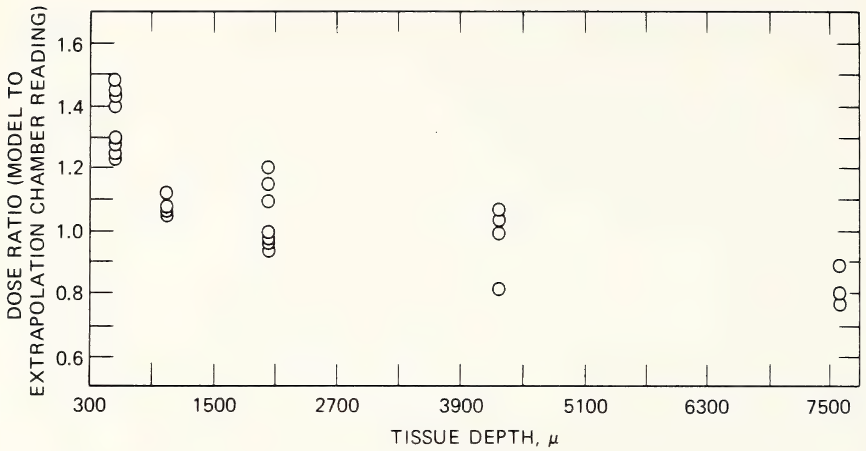

Fig. 4 Ratio of model (TDD) dose to extrapolation-chamber dose, as a function of tissue depth (236-μ particles).

tissue surrounding the point of interest) at a point 100 μ deep "underneath" the source (fallout particle) is termed the "point depth dose" at 100 μ.

For a considerable period of time, beta-radiation damage to skin was viewed almost entirely in terms of the estimated 100-μ point depth dose. However, in recent years it has become generally accepted that for a serious radiation lesion to occur the germinal cells must be destroyed over an area of skin too large for normal regeneration to replace them within a reasonable period of time. Of necessity this has led to consideration of area dose absorption rather than dose absorbed at a specific point.

A survey by Krebs[10] in 1967 showed that, for an acute lesion of the skin to develop, the viable germinal cells must be reduced to a survival level of less than 0.001 over an area sufficiently large to prevent replacement of dead cells via cell proliferation in the margin of the exposure field. The criterion recommended by Krebs is that a 1500-rad or greater dose to the skin, deposited on the periphery of a 4-mm-radius circular field 100 μ deep in tissue, constitutes a potential skin-damage threat.

Krebs derived his conclusions from X-ray microbeam studies. At the time of his evaluation, few biological-damage data were available from single-particle investigations. After Kreb's conclusions were published, an experimental study testing the suggested criterion was conducted.[11] Irradiated microspheres were used as radiation sources, and swine were the experimental animals. Results obtained in this study showed that the minimum radiation dose, deposited at the periphery of a 4-mm-radius field, required to produce a very small ulcer (less than 0.5 mm in diameter) is estimated to be below 405 rads. An ulcer 1 mm in diameter was produced by absorption of 660 rads (same field), a 2-mm-diameter ulcer by about 1150 rads, etc. If we assume linearity of the ulcer diameter with dose (4-mm-radius field), as indicated by the data, then by extrapolation a 350-rad delivery would be sufficient to yield a zero-diameter ulcer.

In this work the 660-rad dose was used as the threshold dose for damage to human skin from deposited fallout particles. This admittedly arbitrary threshold was chosen on the basis that a 1-mm-diameter ulcer is small enough to be considered a threshold for damage but large enough to be recognizable. Choice of 350 or 1150 rads as a threshold dose does not appreciably affect the conclusions derived.

THE MULTIPLE-PARTICLE BETA-DOSE MODEL

The multiple-particle beta-dose model is designed for evaluation of dose situations in which the fallout-particle deposition density on the skin is of such magnitude that beta radiation emitted from adjacent particles is absorbed in the same tissue volume.

Two distinct approaches can be used to examine the absorbed dose from multiparticle sources. In the first the source is viewed as a uniform plane source of strength dependent only on the number of "equivalent fissions" of fission products deposited per unit area. In the more realistic second approach, the source is taken to be a group of fallout particles of size distribution dependent on the weapon yield and the distance from ground zero to the deposition point of interest. The beta dose delivered by such a source to the skin depends, in addition to the particle-size distribution, on the fallout mass deposited per unit area and on the specific activity of the fallout.

The plane-source approach was pursued by Brown,[12] who used Spencer's plane-source calculations to compute beta-dose-rate multipliers for each fission-product beta emitter. Brown considered two situations: (1) contact dose, where the plane source lies between an absorbing medium and a backscatterer, and (2) beta bath, where an attenuation medium separates the absorbing medium from the plane source.

Using Brown's contact-dose multipliers and the output from the abundance code (Code 1) of the TDD program, we can calculate the dose delivered to the skin from a plane source of the desired activity level. Results of these computations are considered later.

In the second, or particulate, model, the source is viewed, for purposes of analytical examination, as consisting of superimposed strata of fallout particles, each stratum being in contact with the skin surface. Each stratum consists of an array of equal-size particles with separate particles placed at the intersections of a uniform rectangular-plane grid. Figure 5 illustrates the concept. The dose is estimated at point X, 100 μ below the central point of the grid plane. The dose at X can be determined by summation of the dose contributions from individual particles as computed by the TDD model.

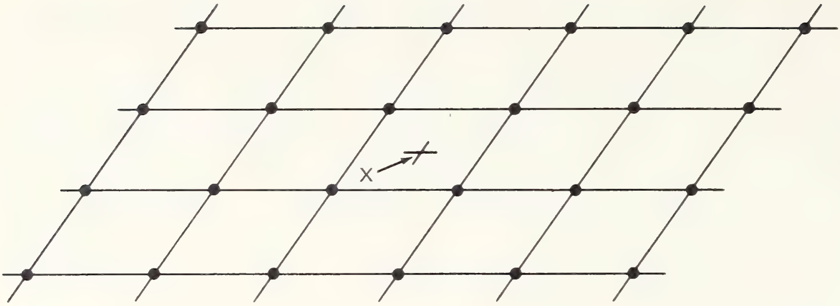

Fig. 5 Schematic of the multiple-particle array concept.

Dimensions of the unit cell of the grid are determined by the mass deposition density (in grams of fallout per square foot) and the size class of particles forming the grid. For a relatively large array of closely spaced particles, the dose at any point 100 μ below the plane becomes very close to the dose at X.

For calculation of the dose at X, dose contributions from the particles closest to X are computed and added. Then doses from particles at increasing distances from X are added until the incremental increase in dose falls below a predetermined fraction of the initial sum, at which time the calculation stops.

For accuracy, 10 strata of arrays were considered in the calculations. Each stratum was assumed to contain 10% of the total fallout mass deposited (on a unit-area basis). Particle sizes for the arrays were determined by the following procedure:

1. Assume a mean and a maximum particle size for the fallout deposit. In the first four situations considered, take the means parametrically as 100, 250, 500, and 700 μ each with a fixed maximum of 1000 μ. In a fifth case take the mean as 1000 and the maximum as 2000 μ.
2. Assuming a log-normal distribution[4] of particle sizes in each case, and with the knowledge of the maximum and the mean, trace a log-probability line for the particle-size distribution.
3. Subdivide the line into 10 equal-probability regions and determine for each region the particle size, corresponding to the midrange probability. Use these 10 mean particle sizes for the strata.

Two facts are worth mentioning here. (1) For obvious reasons, the particulate approach is much more realistic than the plane-source approach. (2) For the same number of equivalent fissions per unit area, the plane-source computations give dose values higher than the Multiparticle Model by as much as an order of magnitude (see Fig. 6). The discrepancies are apparently chiefly due to attenuation within particles. The detailed differences between the dose values resulting from the two approaches depend on the particle-size distribution

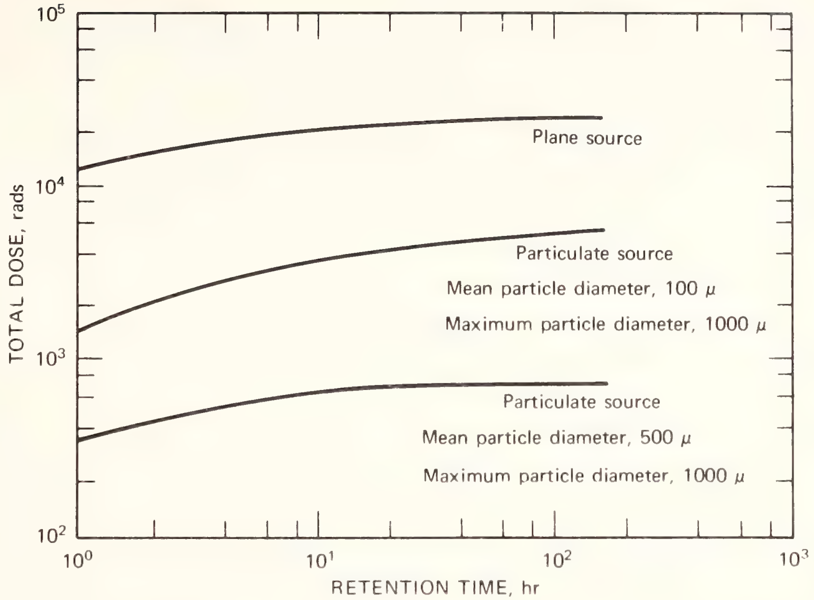

Fig. 6 Comparison between doses computed for a plane source and the corresponding values for a multiparticle source. Tissue depth, 100 μ; delay time, 10^3 sec; deposition density, 100 mg/sq ft; activity, 10^{15} fissions/cc.

assumed in the particulate approach (Fig. 6). For a fixed maximum size, the difference decreases as the mean particle size decreases, but a factor of 5 was the smallest encountered for the cases considered.

DOSE CRITERIA FOR MULTIPLE-PARTICLE DEPOSITION

To date no criterion has been explicitly proposed for skin damage from multiple particles. However, the following points serve as guidelines for establishing such a criterion:

1. As in the case of single-particle sources, damage to the skin will occur when the survival level of the germinative cells is reduced to less than 0.001 over an area sufficiently large to preclude replacement of dead cells via proliferation.[10]

2. Such a reduction in survival occurs at a lower dose level from a multiparticle source than from a single-particle source. Krebs estimates that a uniform 1300-rad dose from a multiparticle source would cause the same reduction in survival level brought about by a 1500-rad dose from a single-particle source.[13]

3. In view of the difference between the predicted single-particle critical dose (1500 rads) and the corresponding experimentally determined value of 660 rads, an adjustment has to be made to the suggested multiple-particle value to bring it into line with experiment.

4. It seems reasonable to accept a proportional dose for the multiparticle situation; i.e., $(1300/1500) \times 660 \approx 570$ rads. That is, exposure of the skin (100-μ depth) to a uniform deposited dose of 570 rads from a multiple source will be assumed sufficient to damage the skin in the manner described for the single-particle exposure.

RESULTS AND DISCUSSION

Doses from Single Particles

Point depth doses (estimated at 100-μ tissue depth directly below the fallout particle) and Krebs doses (estimated at a point radially displaced 4000 μ in a plane 100 μ below the skin surface) were computed for particles 50, 100, 200, 500, 750, and 1000 μ in diameter; for each particle size, doses were computed for 10^3, 10^4, 10^5, and 10^6 sec of delay time (time between weapon detonation and deposition of the particle on the skin). The fallout particles were assumed to contain 10^{15} fissions per cubic centimeter. For all but exceptional situations, 10^{15} fissions/cc is considered the maximum expected fallout activity. Beta doses from fallout of higher fission density can be obtained from the values reported here by linear extrapolation.

Figures 7 to 10 present samples of the computer-plotted doses as functions of particle retention time on the skin. It can be seen from Fig. 7, which presents Krebs doses for the earliest particle arrival time considered, that single fallout particles smaller than 500 μ in diameter, landing on the skin as early as 10^3 sec (16.7 min) after detonation, will not cause any skin burns. A single 500-μ particle arriving even this early has to be retained about 10 hr before it delivers the 660 rads required for damage. Table 1 shows experimental data obtained at Oak Ridge National Laboratory (ORNL) for expected retention times of particles on human skin under normal conditions of temperature and humidity.[14] Considering the values in Table 1, even a 500-μ particle would obviously be incapable of producing a 1-mm lesion.

Figure 8 presents the point depth doses delivered by the same particles under the same (early arrival) conditions. Comparison of Figs. 7 and 8 shows that point depth doses are higher than the corresponding Krebs doses by a factor of 10^2 to 10^3 depending on the particle size. Lower ratios correspond to larger particle sizes.

From Figs. 9 and 10, it can be seen that, after a delay of a little over 10^4 sec (about 2.8 hr), even a 1000-μ particle can be tolerated, provided its retention time does not exceed its expected value in Table 1.

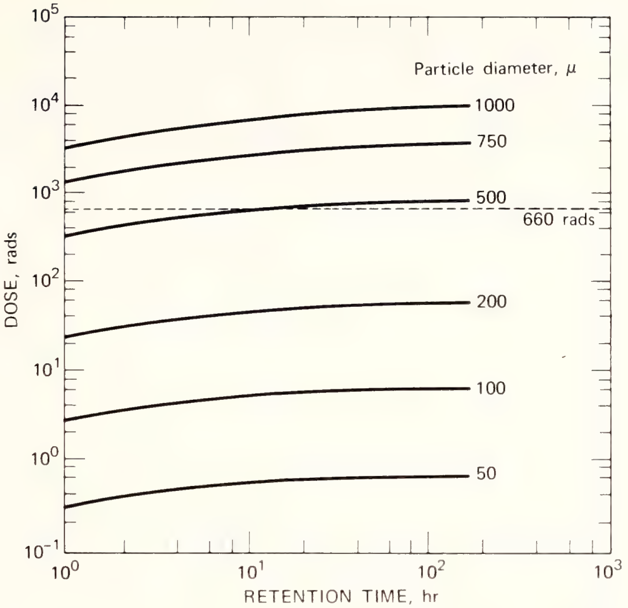

Fig. 7 Krebs dose delivered to the skin by single fallout particles at an exposure starting time of 10^3 sec after detonation. Tissue depth, 100 μ.

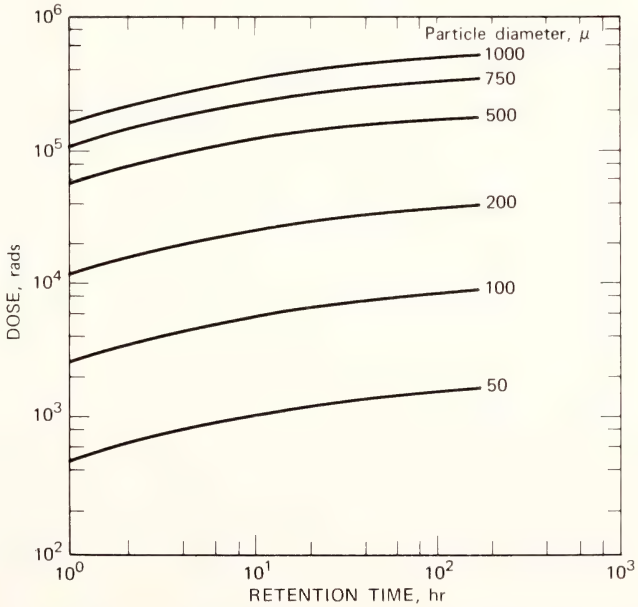

Fig. 8 Point depth dose delivered to the skin by single fallout particles at an exposure starting time of 10^3 sec after detonation. Tissue depth, 100 μ.

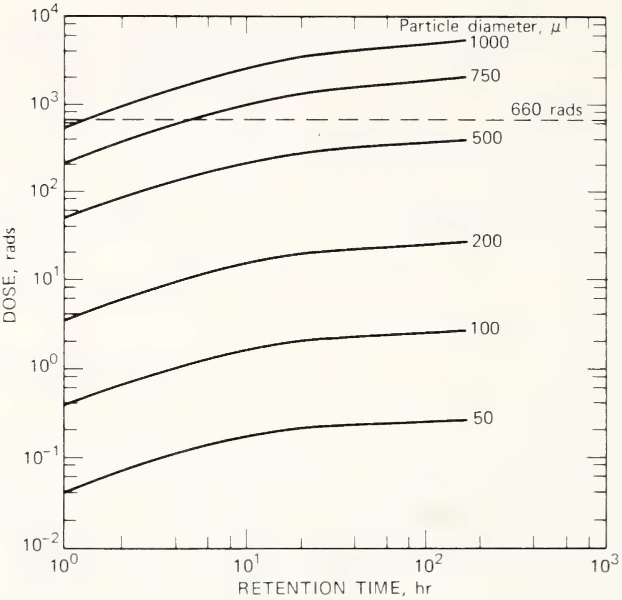

Fig. 9 Krebs dose delivered to the skin by single fallout particles at an exposure starting time of 10^4 sec after detonation. Tissue depth, 100 μ.

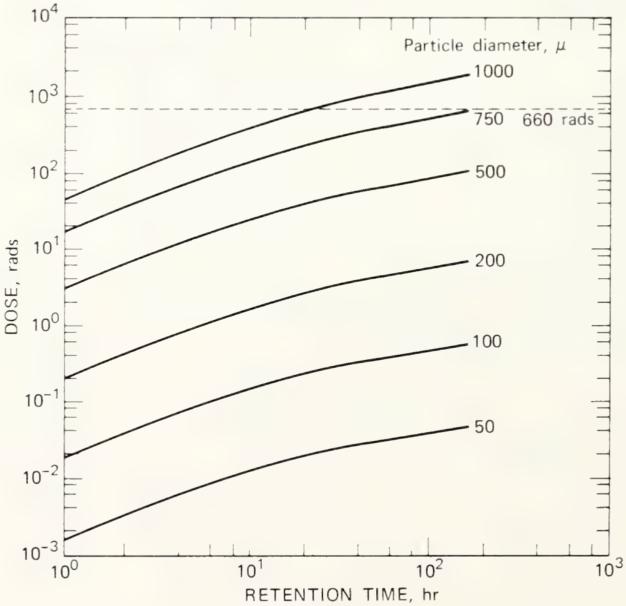

Fig. 10 Krebs dose delivered to the skin by single fallout particles at an exposure starting time of 10^5 sec after detonation. Tissue depth, 100 μ.

Table 1

EXPECTED RETENTION TIMES OF PARTICLES ON HUMAN SKIN*

Particle diameter, μ	Time, hr
50	6.8
100	3.5
200	2.7
500	2.2
750	2.1
1000	2.0

*From Ref. 14.

Figure 10 shows further that, after a delay of 10^5 sec (about 28 hr), no single particle of any size can possibly cause a beta burn (except for the 1000-μ particle retained for an inordinately long time).

Doses from Multiparticle Fallout

Samples of the data computed with the Multiparticle Model are shown in Figs. 11 to 15. In these figures time-integrated doses from fallout deposition densities of 100, 200, 500, 1000, 2000, and 5000 mg/sq ft for different particle-size distributions have been plotted as functions of fallout retention time. All computations are based on 10^{15} fissions/cc. Delay times of 10^3, 10^4, 10^5, and 10^6 sec are covered.

Figure 11 shows that for a delay time of 10^3 sec even the lowest deposition density (100 mg/sq ft) of particles of 100-μ mean diameter and 1000-μ maximum diameter (size distribution A) can deliver to the skin in less than 1 hr more than the 570 rads required for damage in the multiparticle situation. However, as seen in Fig. 12, the same mass of fallout of 1000-μ mean diameter and 2000-μ maximum diameter (size distribution B) delivers a maximum of only 300 rads, even if retained over 100 hr. Other size distributions give intermediate doses.

The situation changes somewhat at the next higher fallout-arrival (delay) time, 10^4 sec. A 200 mg/sq ft deposit of size distribution A can be tolerated in this case for about 1.5 hr (Fig. 13).

After a delay of 10^5 sec, a 2000 mg/sq ft deposit of size distribution A gives the critical 570 rads in about 1.5 hr (Fig. 14); after a delay of 10^6 sec (11.5 days), it takes 5000 mg/sq ft of the same size distribution about 10 hr to cause skin burns (Fig. 15).

Other formulations of output data can be derived from the multiparticle dose computations. A few examples follow.

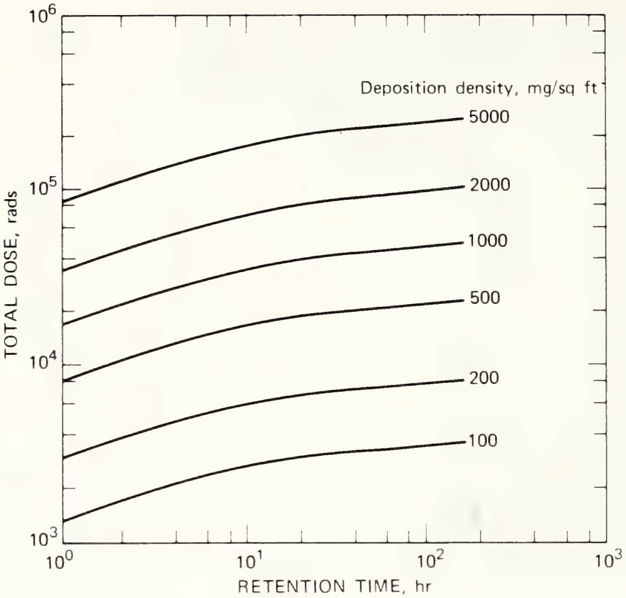

Fig. 11 Dose delivered to the skin by multiparticle fallout of 100-μ mean diameter and 1000-μ maximum diameter at an exposure starting time of 10^3 sec after detonation. Tissue depth, 100 μ.

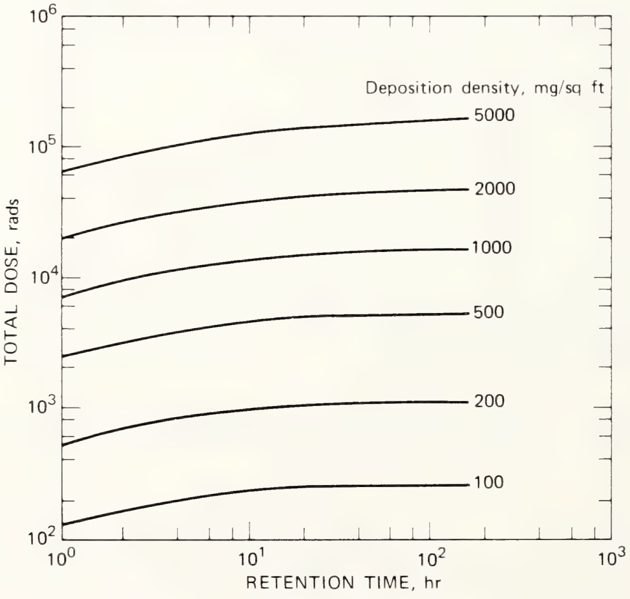

Fig. 12 Dose delivered to the skin by multiparticle fallout of 1000-μ mean diameter and 2000-μ maximum diameter at an exposure starting time of 10^3 sec after detonation. Tissue depth, 100 μ.

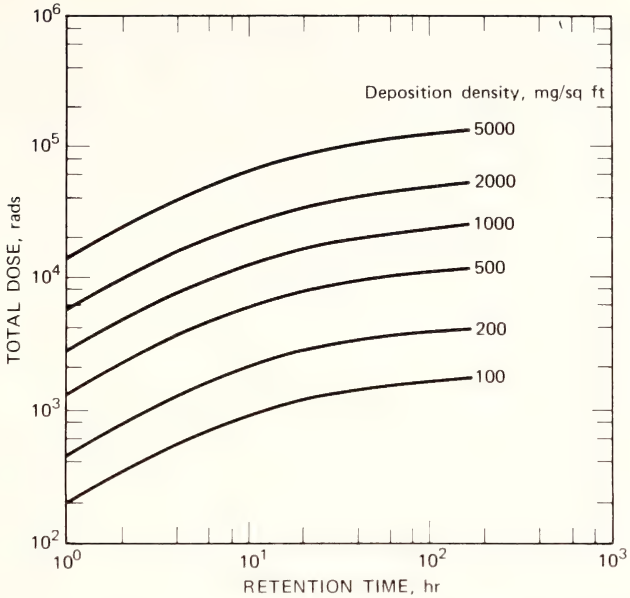

Fig. 13 Dose delivered to the skin by multiparticle fallout of 100-μ mean diameter and 1000-μ maximum diameter at an exposure starting time of 10^4 sec after detonation. Tissue depth, 100 μ.

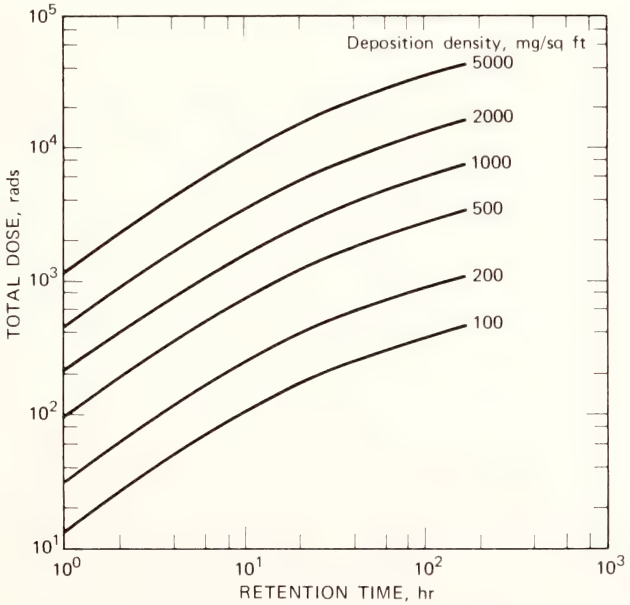

Fig. 14 Dose delivered to the skin by multiparticle fallout of 100-μ mean diameter and 1000-μ maximum diameter at an exposure starting time of 10^5 sec after detonation. Tissue depth, 100 μ.

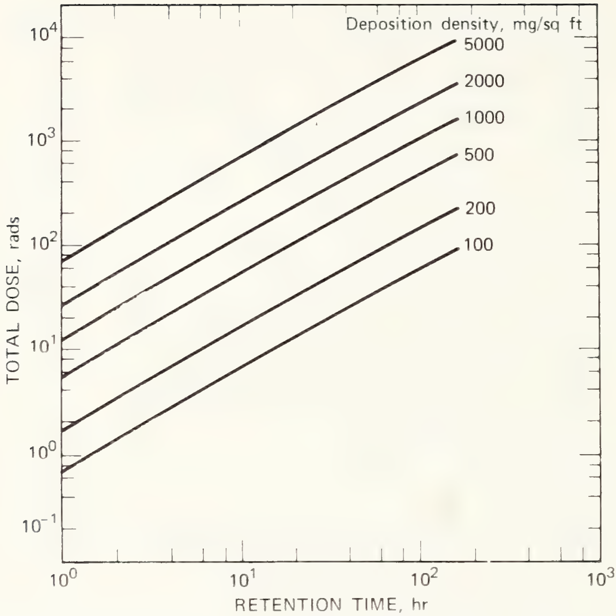

Fig. 15 Doses delivered to the skin by multiparticle fallout of 100-μ mean diameter and 1000-μ maximum diameter at an exposure starting time of 10^6 sec after detonation. Tissue depth, 100 μ.

Table 2 presents one formulation, the effect of exposure-initiation time (delay time) on the Krebs dose received by the skin from the same fallout deposition density. The table presents doses delivered by two deposition densities, 100 and 2000 mg/sq ft, in each case over 1-, 2-, 5-, 10-, and 24-hr exposure periods, all following delays of 24, 48, 72, and 168 hr. Also given are the time periods for which fallout under these conditions would have to be retained before delivery of 570 rads takes place if the exposure starts at 24, 48, 72, and 168 hr postdetonation. In both parts of the table, size distribution A is assumed.

Another type of output formulation that may be useful (not illustrated) would show the skin dose accumulated in 1 hr, e.g., starting at fallout arrival or some later time, as a function of distance from ground zero for various weapon yields. The figure could be obtained by combining the dose data given here with the data of Clark and Cobbin,[15] for example; the latter data relate midrange particle size to downwind distance from ground zero for different weapon yields. It must be recognized that, for a given weapon yield and downwind distance, fallout phenomenology, as exemplified by the Clark–Cobbin approach, specifies uniquely not only (1) the midrange particle size but also (2) the ground-surface deposition density and (3) the times of fallout arrival and

Table 2

EFFECT OF EXPOSURE-INITIATION TIME ON KREBS DOSES DELIVERED TO THE SKIN*

Retention time, hr	Exposure-initiation time 24 hr	48 hr	72 hr	168 hr
Fallout Deposition Density of 100 mg/sq ft†				
	Doses received, rads:			
1	18	8	5	2
2	36	16	10	4
5	84	39	24	9
10	151	73	46	17
24	291	153	101	41
	Retention times required to accumulate 570 rads, hr:			
	76	250	600	2400
Fallout Deposition Density of 2000 mg/sq ft‡				
	Doses received, rads:			
1	466	217	120	50
2	912	423	230	98
5	2138	998	608	228
10	3862	1879	1182	445
24	7420	3918	2571	1030
	Retention times required to accumulate 570 rads:			
	78 min	170 min	4 hr, 40 min	13 hr, 20 min

*Mean particle diameter of 100 μ and maximum particle diameter of 1000 μ.

†4×10^{13} fissions/sq ft.

‡8×10^{14} fissions/sq ft.

cessation. The unique values of the deposition density and times of arrival and cessation would have to be considered in the preparation of a family of curves covering a range of weapon yields. Skin deposition density could be parameterized at, for example, 100 mg/sq ft, which would allow for consideration of fallout-particle resuspension, or simply for normalization to the correct skin-deposition value at each point. A carefully planned family of curves could thus provide a picture of those yields and downwind distances at which a 1-hr exposure to fallout which starts to deposit on the skin at arrival time or later would produce the critical skin dose of 570 rads. Such kinds of results could be most useful in postattack planning.

REFERENCES

1. R. Loevinger, E. M. Japha, and G. C. Brownell, Discrete Radioisotope Sources, in *Radiation Dosimetry*, pp. 693–754, Academic Press, Inc., New York, 1956.
2. J. C. Ulberg and D. B. Kochendorfer, Models for Estimating Beta Dose to Tissue from Particle Debris in Aerospace Nuclear Applications, USAEC Report USNRDL-TR-1107, Naval Radiological Defense Laboratory, Dec. 12, 1966.
3. O. H. Hogan, P. E. Zigman, and J. L. Mackin, Beta Spectra II. Spectra of Individual Negatron Emitters, Naval Radiological Defense Laboratory, USAEC Report USNRDL-TR-802, Dec. 16, 1964.
4. C. F. Miller, Fallout and Radiological Countermeasures. Volume I, Report AD-410522, Stanford Research Institute (Project No. IM-4021), 1963.
5. R. F. Korts and J. H. Norman, A Calculational Model for Condensed State Diffusion Controlled Fission Product Absorption During Fallout Formation, Report GA-7598, General Dynamics Corp., General Atomic Div., Jan. 10, 1967.
6. L. V. Spencer, Energy Dissipation by Fast Electrons, National Bureau of Standards, Monograph 1, 1959.
7. G. B. Curtis and J. S. Petty, A Program to Compute Beta Radiation Dosage. Volume I. Program Description. Final Report, USAEC Report TID-24033(Vol. 1), American Research Corp., Aug. 1, 1967.
8. S. Z. Mikhail, Tissue Beta Radiation Doses from Particulate Fission-Product Sources: Comparison of Model Predictions with Experimental and Monte Carlo Values, Report ESA-TR-70-01, Environmental Science Associates, 1970.
9. R. Loevinger, Extrapolation Chamber for the Measurement of Beta Sources, *Rev. Sci. Instr.*, **24**: 907 (1953).
10. J. S. Krebs, The Response of Mammalian Skin to Irradiation with Particles of Reactor Debris, Report USNRDL-TR-67-118, Naval Radiological Defense Laboratory, 1967.
11. P. D. Forbes and S. Z. Mikhail, Acute Lesions in Skin Produced by Radioactive Microspheres, Temple University Report, Contract SNPN-49, 1970.
12. S. L. Brown, Disintegration Rate Multipliers in Beta-Emitter Dose Calculations, USAEC file No. NP-15714, Stanford Research Institute (SRI Project MU-5116), August 1965.
13. J. S. Krebs,Stanford Research Institute, personal communication, September 1970.
14. B. R. Fish, Environmental Studies: Radiological Significance of Nuclear Rocket Debris. Semiannual Progress Report July 1–Dec. 31, 1964, USAEC Report ORNL-TM-1053, Oak Ridge National Laboratory, April 1965. (Classified)
15. D. E. Clark, Jr., and W. C. Cobbin, Some Relationships Among Particle Size, Mass Level and Radiation Intensity of Fallout from a Land-Surface Nuclear Detonation, Report USNRDL-TR-639, Naval Radiological Defense Laboratory, Mar. 21, 1963.

PROPERTIES OF FALLOUT IMPORTANT TO AGRICULTURE

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ABSTRACT

The intrinsic properties of fallout associated with radiological hazards which could affect agricultural operations in the postattack period of a nuclear war include: (1) the radionuclide composition of the fallout material, which determines the energy composition of the gamma and beta radiation emitted, (2) the physical and chemical properties of the fallout particles (such as size, shape, composition, structure, and solubility) which influence their retention by surfaces, and (3) the solubility and biological availability of specific radionuclides. In terms of crop or agricultural-product output, both operational factors (effects on man and his social system) and biological factors (response of plants and animals) would be important.

Since the degree of the hazard to the food-producing agricultural systems would generally depend more on external parameters (such as the available weapon system, form or mode of attack, level of attack, explosive yield of weapons, relative heights of burst, and local and regional weather patterns) than on the properties of the fallout, these parameters are discussed in detail.

A major recent development in weapon systems which could have a significant impact on the type and extent of hazard to agriculture in a nuclear war is the Multiple Independent Targeted Reentry Vehicle (MIRV). Estimates of MIRV system capabilities, especially in terms of using many smaller-yield warheads on many smaller targets, are used to identify several important implications for future civil-defense planning and the role of civil-defense capabilities in the relative deterrence posture. If sufficient fallout shelters with protection factors of 130 or more were available for the U. S. population, it appears that the U.S.S.R. could not deploy sufficient SS-9 missiles to assure the destruction of the U. S. population within the next 800 years (even with MIRV) using currently available technology. Also, the effect of MIRV and the associated lower-yield warheads would be to almost eliminate the widespread fallout effects previously estimated for attacks in which land-surface detonations of weapons in the megaton-yield range have been postulated; a comparable degree of effect on agriculture might be achieved from attacks that are designed to kill more than 65% of the U. S. population if all detonations in rural areas were surface bursts.

The more important properties of fallout which could significantly affect agricultural operations after a nuclear war are those related to the total gamma- and beta-radiation emissions from the particles and the physical and chemical properties of the particles which influence their retention by plant and animal surfaces. In addition, physiochemical properties of the fallout, such as the solubility of individual radionuclides, can become important radiological-hazard problems in the production and consumption of specific agricultural products. A well-publicized example of this is the relative solubility of ^{131}I and its accumulation in milk produced by cows that have ingested fallout-contaminated food and water.

In terms of radiobiological effects to agriculturally important plants and animals, previous analyses have shown that the major cause of radiation damage would be the exposure of temporal units of the biota of rural farmland ecosystems to ionizing radiation.[1] Under the subject of longer-term ecological effects, the major concern would be with the secondary effects to functional units of the biosphere including biological populations, communities, and ecosystems.[2] Secondary effects, in contrast to direct effects, are disturbances and injury or damage, usually caused by the direct effects, which do not become important or do not develop until some time later. One property of fallout that could affect the relative severity of short-term and long-term effects on agricultural plants and animals is the combined decay rate of the radionuclides in the fallout; another property is the energy spectrum of the absorbed radiation.

SOURCE OF RADIOLOGICAL INJURY OR DAMAGE

In a nuclear explosion more than a hundred radioactive fission-product nuclides and many additional neutron-induced radionuclides are produced. This radioactive mixture initially consists of radionuclides with radioactivity-decay half-life values that vary from a fraction of a second to many years. Since most of the radionuclides emit both beta particles and gamma rays when they disintegrate, these two types of ionizing radiation are present in a fallout environment as potential causes of biological damage to living tissue. The presence of all these radionuclides in an ecosystem thus constitutes a source of radiological hazard from fallout. The major radiological hazard to man is external gamma radiation from deposited fallout; this fact requires special recognition both in damage-assessment studies and in civil-defense planning.

Fallout particles from land-surface detonations as nuclear-radiation sources consist of fused, sintered, and unchanged grains of soil minerals or other materials present at the point of detonation.[3] Also present to a minor extent in fallout particles are inert materials from the weapon or warhead, as well as the radioactive elements produced in the fission and neutron-capture processes occurring at detonation. Roughly, the relative amounts of soil minerals, bomb-construction materials, and radioactive elements in fallout particles are

(1) up to 1 Mt of soil per megaton of total weapon yield, (2) of the order of 1 ton of warhead materials per megaton of total weapon yield, (3) about 120 lb of fission products per megaton of fission yield, and (4) about 100 to 200 lb of induced radioactive atoms per megaton of total yield.

Analyses of fallout particles from surface and near-surface detonations collected at weapons tests at both the Eniwetok Proving Ground and the Nevada Test Site show that the radioactive elements are either within the interior of fused and sintered particles or are attached to the exterior layers of all three types of particles.[3] Larger fallout particles are formed, not by the condensation of vaporized soil, but from individual or agglomerated soil particles that originally either existed as single soil grains or were produced through the breakup of a fused mass of liquid soil or rock. All three types of particles are drawn into the rising fireball and apparently serve as collectors for small vapor-condensed particles and as condensation centers for vaporized fission-product and radioactive neutron-induced atoms.

On the basis of physiochemical properties of common metallic oxides of the chemical elements in soil and coral, it can be concluded that the fallout-formation process does not begin until the fireball temperature (or the temperature of the gaseous material in the fireball) has decreased to about 3000°K, because at higher temperatures all materials tend to dissociate rather than to condense. As the fireball temperature decreases below about 3000°K, vapor-condensation processes should take place to produce very small liquid particles. Such small particles have been observed in worldwide fallout collections and as attached particles on unchanged coral grains in the fallout materials collected from weapons tests at the Eniwetok Proving Ground.

As the fireball rises and cools and the crater materials are drawn up into its volume, the thermal action at the surfaces of entering molten particles should gradually change from a vaporization process to a condensation process in which the less volatile fission products condense onto and diffuse into the liquid phase of the particles. In addition, the larger molten soil particles, as they circulate through the fireball volume, would rapidly form agglomerates with a large fraction of the smaller, previously formed, vapor-condensed particles. Particles entering the fireball volume at later times may be heated to sintering temperature or may never be thermally altered.

As the surface temperature of the particles decreases, the rate of diffusion of the condensed radioactive atoms into the interiors of the particles should also decrease so that the more volatile of the radioactive elements, which can condense only at lower temperatures, collect and concentrate on the exterior surface of the particles. Also, radioactive daughter atoms (even if not volatile) formed at later times from volatile parent nuclides, such as those from rare-gas elements, would be concentrated on the exteriors of the smaller particles. Because of the differences in volatility as a function of temperature among the various fission-product elements, fractional condensation would be expected to occur throughout the whole fallout-formation process. The observed degree of solubility and biological availability of such radionuclides as ^{89}Sr, ^{90}Sr, and ^{137}Cs from the fallout of nuclear-weapons tests strongly supports these views regarding the condensation process.[4]

In general, two rather distinct periods of fallout formation by condensation processes have been postulated.[3] In the first period the condensation of volatile radioelements is considered to occur by deposition onto and diffusion into large molten soil particles and by agglomeration with smaller particles. The radioelements thus condensed would become fused within the volumes of the molten particles when they cooled and solidified. In the second period the remaining volatile gaseous radioelements condense onto the surfaces of relatively cold solid particles (most of which consist of late-entering, thermally unaltered grains of soil).

The significant chemical property associated with the amount of a radioelement that condenses during the second period of formation is its potential solubility, whereby it can become biologically available for later assimilation by plants and animals. The more volatile radioelements in fallout are more soluble and more biologically available than the refractory elements. However, the fractional degree to which each element condenses in either period of condensation is expected to depend very much on both the temperature and the rate of temperature decrease, which determine the conditions and times at which diffusion into the particle effectively ceases and at which the condensing radioelement begins to concentrate on the surface of the particles.

If all the materials produced in a land-surface nuclear detonation and all entering the fireball volume remained together for the first 5 or 10 min after detonation, the radioactive composition and the subsequent radioactive decay (and nuclide solubility) would be about the same for all fallout particles. However, it is known that all the entering particles do not remain together in the fireball and cloud for such periods of time. Immediately after the fireball expands to maximum size it begins to rise in the air. The upward movement of the hot gases sets in motion a large-scale toroidal circulation because of the drag forces of the surrounding air. This toroidal motion, with circulation velocities in excess of 100 mph, is probably responsible for setting up air motions whose forces are sufficiently strong to pull the blast-loosened soil from the crater and crater lip into the rising fireball.

The circulation of the particles in the toroid should result in rapid separation of the larger particles from the circulating mass of condensing gases and should, by centrifugal force, move them to the periphery of the toroid. When the circulating particles reach the periphery or the bottom of the cloud and the pull of gravity begins to exceed the upward drag forces of the air near the base of the rising cloud, the particles should begin falling toward the earth. Other particles of the same size that are not yet near the periphery of the toroid may continue to circulate for a much longer time before they leave the base of the cloud.

These views of particle circulation and formation are suggested by (1) the relatively long period over which particles of a given size arrive on the ground, (2) the relatively early initial arrival times for close-in fallout, (3) the variation in composition of the radioelements carried by particles of different sizes, and (4) the variation in specific activity and radioelement composition among particles of a given size.

The concentration of the volatile radioelements in the radioactive compositions carried by the larger particles is generally low. This relatively low concentration could occur only through the earlier ejection of the large particles from the volume of the fireball containing the radioelements (vapors plus small vapor-condensed particles). In addition, the large fallout particles from many low tower detonations do not contain or carry any soluble radioelements; therefore these particles must have been ejected from the rising fireball or cloud when the particle surfaces were still at a very high temperature. Thus the toroidal motion is considered to be partially responsible for the observed differences in gross radioactive decay and biological availability of different radioelements carried by fallout particles of different diameters.

The toroidal motion, which apparently causes early ejection of the larger particles (i.e., early with respect to time-of-fall from the height of the stabilized cloud), can also cause prolonged apparent buoyancy of the smaller particles. The smaller particles should circulate for longer times and should remain in the toroid where they could adsorb the more volatile radioactive elements on their surfaces. Essentially all fallout particles, except those with diameters less than about 50 to 80 μ, apparently leave the cloud volume under influences of toroidal circulation.

No observed data exist on the properties of fallout from detonations on soils similar to those of likely targets in a nuclear war. In fact, only a few detonations at the Eniwetok Proving Ground and the Nevada Test Site have provided data useful for the development of fallout models for land-surface detonations. All the large-yield test devices were detonated over water, on coral atolls, or in the air. Most test detonations in the yield range of 1 kt to 1 Mt were mounted on towers. Consequently there is no evidence proving that all types of fallout information obtained from the weapons tests (even under suitable detonation conditions) are satisfactory for evaluating computational procedures developed to give quantitative estimates of properties of the fallout particles, as well as of their distribution over the country as a consequence of an assumed set of nuclear detonations on specified targets in the continential United States. Further theoretical developments and supporting experimental work are needed to evaluate and improve the validity of some available input data used in the formulation of many fallout models.

The radionuclides in worldwide fallout from high airbursts, in contrast to those described for the close-in local fallout from near-surface detonations, are generally quite soluble. Therefore essentially all the radionuclides in long-range worldwide fallout are biologically available. Fused-type particles formed from the warhead or bomb materials have been identified and found in fairly large numbers in stratospheric collections of bomb debris.[5] But a large fraction of the worldwide fallout from a large-yield nuclear airburst is apparently formed in the stratosphere at some time after detonation through processes of coagulation and coprecipitation of the radioactive atoms with the natural stratospheric aerosol particles. These particles, composed mainly of water-soluble ammonium sulfate compounds, apparently serve as carriers for eventually returning the longer-lived radioactive elements to earth.

Under all conditions of detonation that lead to the production of fallout, the form and properties of the fallout particles are determined during the cooling period of the fireball and cloud; for the decay products of gaseous and several other radioelements, the attachment to particles occurs at later times. The materials in or entering the fireball at these times are particularly important factors in determining the properties of the fallout particles. These formation processes set the stage for all subsequent radiological interactions between the fallout materials and the biological and ecological environments in which they deposit.

One of the chief difficulties in predicting fallout levels at a given location, in addition to the problem of defining the fallout-particle-cloud source, lies in the problems associated with analyzing and predicting the wind fields. The winds at all altitudes through which the particles fall, of course, determine how the fallout particles are distributed over the earth's surface. Other major factors for which very little accurate data exist, especially for fallout from detonations over silicate soils, include (1) variation of the specific activity of fallout with particle size and (2) influence of weapon yield, burst height, and environmental material (soils and other likely target materials) on the gross particle-size distribution of the fallout (i.e., by particle number, mass, or radioactivity content).

The radiation, chemical, and physical properties resulting from the fallout-formation processes and conditions may give rise to one or more of five major types of radiological hazard to biological species. These are (1) external gamma hazard, as mentioned for humans, (2) contact beta hazard, (3) beta-field hazard, (4) internal hazard from ingested radionuclides, and (5) inhalation hazard.

The nature of the hazard and the response of biological species to it are perhaps better known and understood for external gamma radiation than for the other four hazards. Under most exposure situations occurring under nuclear war conditions, the external gamma hazard would be the major cause of serious direct radiation injury to large biological species.

The contact beta hazard could arise when fresh fallout particles remained in contact with biological tissue for some period of time. Humans could easily avoid this type of exposure by wiping or brushing fallout particles from exposed skin. This hazard would develop only during and shortly after fallout deposition. After several days the fallout particles would no longer have the radioactive content necessary to cause serious damage to skin tissues. Some data on the retention of particles by humans[6] and on skin doses to animals[7] have been reported. No reliable correlations of such data with fallout-deposition levels have yet been made, but unverified relations between the two have been proposed.[8] A few sets of computations and experimental measurements have been made of the contact beta dose to plants;[9] data on the retention of fallout particles by the foliage of many different types of plants have been reported.[6]

The beta-field hazard (sometimes called the "beta-bath" hazard) could occur in certain confined radiation source geometries for humans. It would be expected to be severe for small plants, small animals, and insects whose habitats become covered with the deposited fallout particles. In such geometries the beta-to-gamma ratio (i.e., the rad-to-roentgen ratio) would generally be between 30 to 100 for fallout-radiation compositions similar to those of past weapons tests. No mathematical models on the beta-field hazard to small plants and animals or insects are known to exist; however, some related work on this hazard was reported.[10,11] The combined radiological hazards (external gamma, contact beta, and beta-field) for plants, animals, and insects should be considered in future research investigations.

The internal hazard from ingested radionuclides and the consequent pattern of exposure of humans, animals, plants, and insects to this hazard after a nuclear war would depend mainly on their uptake and assimilation of biologically available (soluble) radionuclides. Several major processes are involved in the entry of the radionuclides into food chains (or webs). The internal hazard from fallout is characterized mainly by the fact that, at least in humans and other large vertebrate animals, most of the radiation sources (e.g., radioactive atoms) tend to concentrate in specific body organs and that assimilation occurs according to the biochemical properties of specific radionuclides. Thus evaluations of the internal hazard must consider the behavior patterns of each radioelement in the fallout. Data on absorbed doses from ingestion of radionuclides by adult humans have been developed in a significant research effort conducted by Morgan and co-workers[12] over the past 15 years. Similar sets of data for the absorbed doses for young people during their growing years have yet to be developed. Kulp et al.[13] developed a bone model for the uptake of ^{90}Sr in worldwide fallout. Models for estimating the absorbed dose from assimilation of radionuclides in organs of humans have been developed.[14]

The inhalation hazard would be associated with the inhalation and deposition in the respiratory system of small fallout particles of a narrow size range. All the available data on exposure of animals in fallout areas at weapons tests and in laboratories, on air-filter samples in various fallout environments, and on fallout-particle resuspension in air give negligible results for the inhalation hazard. Therefore this hazard is considered to be minor relative to other possible radiological hazards.

The major primary radiological hazards that apparently would cause most damage to farmland (and wild land) ecosystems are external gamma and beta radiation and internal beta radiation from assimilation of radionuclides. It is significant for biological repair and recovery processes that injury sustained from external radiological hazards under nuclear war conditions would generally be more comparable to an acute assault than to a chronic assault, whereas the assimilation of radionuclides would be mainly a chronic exposure to low levels of nuclear radiation. The general effect of radionuclide cycling in species of ecosystems appears from all available data to be mainly a long-term public-health problem rather than a cause of injury leading to the death of biological species.

Because of the large variability in the radiosensitivity of plants according to species, age, and period between growth and reproduction cycles, the gross effects in plant population from exposure to gamma radiation would depend a great deal on the time of year, perhaps of month, when an attack occurred. Thus the total consequence would depend on the targeting pattern for many agricultural areas; the Midwestern states, for example, could receive high levels of fallout from high-yield surface detonations on missile sites in neighboring states and in the Rocky Mountain area.

RADIOLOGICAL DAMAGE ASSESSMENTS

Current Weapons Systems

In most damage-assessement analyses, military targets normally play an important role in establishing the pattern of weapon delivery for any hypothetical attack. For many military targets it is appropriate to assume ground-surface detonations to assure destruction of the target components. Therefore in such attack patterns, called counterforce attacks, a large amount of local fallout is produced. Furthermore, in such studies rather large weapon yields are customarily assigned to military targets, perhaps for consistency with the assured destruction concept.* The relative area of the continental United States within a given standard (H + 1) exposure-rate contour (using an open-field radiation source as the reference condition) as a function of attack level in total megatons detonated is shown in Fig. 1 (Ref. 15). The relative area of the continental United States at a given attack level is shown in Fig. 2 as a function of the standard exposure-rate-contour level.

For hypothetical nuclear attacks on the United States in which most individual weapon yields are assumed (or assigned) to be in the range of 1 to 10 Mt, pure counterforce attacks at attack levels very much larger than 10,000 Mt would not be realistic, at least on a first-strike basis, because of the limit in number of military targets. Thus the extrapolation of the solid lines in Fig. 1 to the higher attack levels probably does not represent any real situation.

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*The assured destruction concept is an extension of the notion that a weapon system or attack pattern can be designed or deduced to perform as envisioned by calculation, within a specified degree of assurance or a stated degree of reliability, ipso facto. The concept is to some degree a technical embellishment evolved by military technicians and analysts to provide a logical basis for deterrence policies.

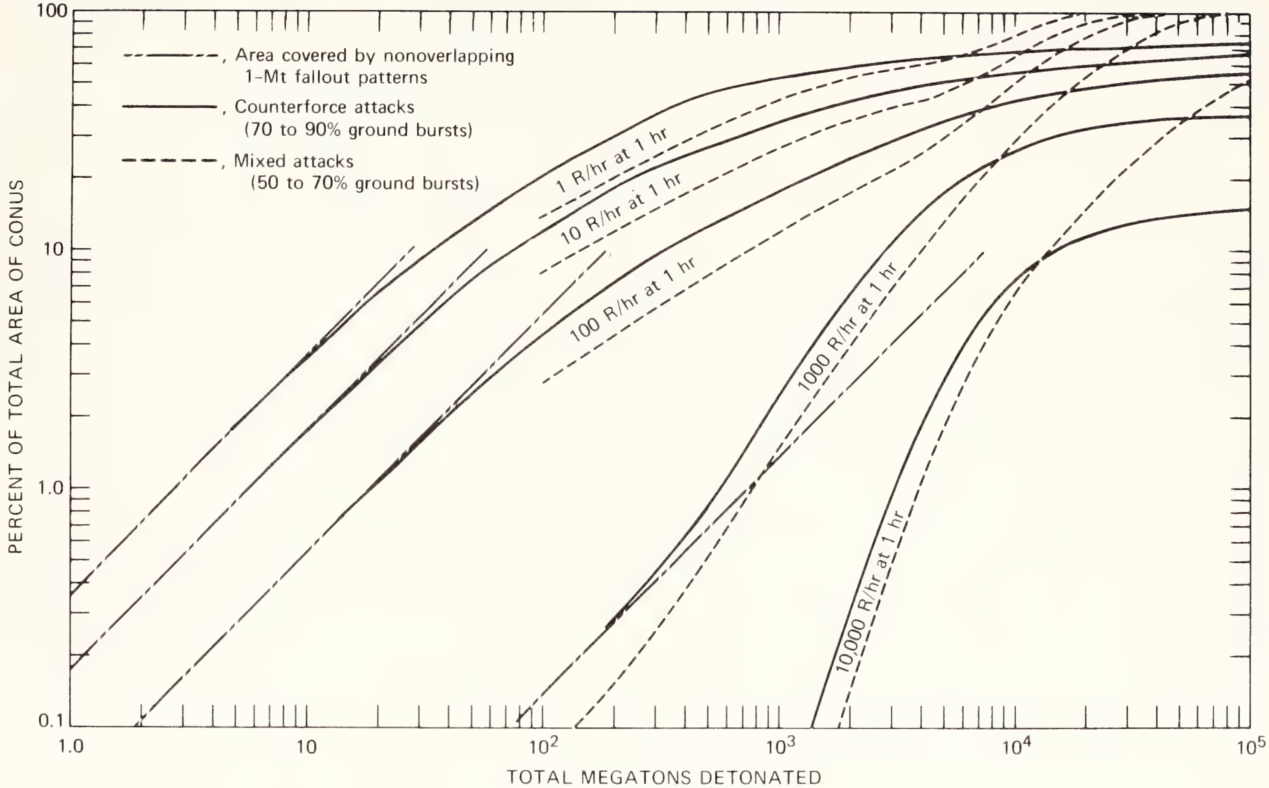

Fig. 1 Percent of area of the continental United States enclosed within selected I_s contours as a function of attack weight (50% fission weapons).

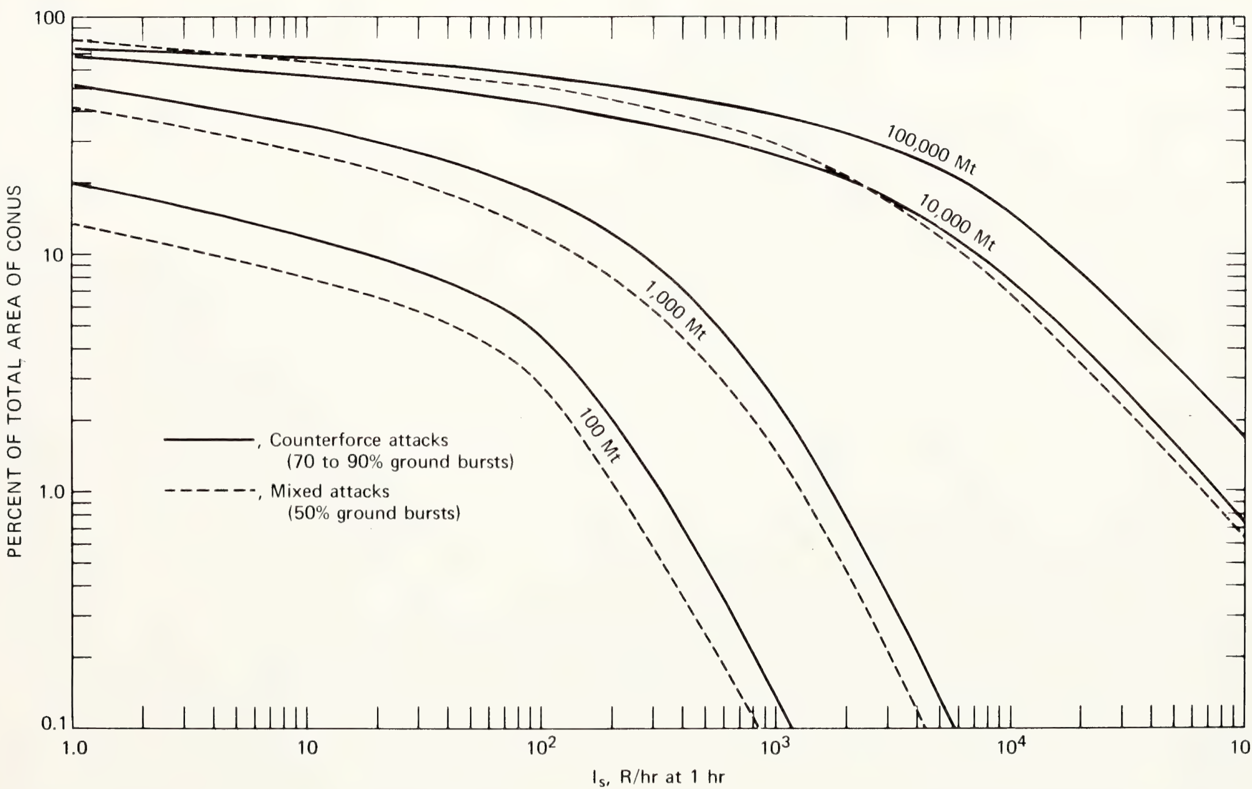

Fig. 2 Percent of area of the continental United States enclosed within I_S contours for selected attack weights (50% fission weapons).

As a general guide, I_s values up to 10 R/hr at 1 hr for fallout from fission weapons do not represent a serious direct radiological hazard to humans or to most other biological species. At I_s values greater than 100 R/hr at 1 hr, extended (but variable) stay times in shelter generally would be required to avoid possible effects of radiation sickness to humans. The I_s values of about 1000 R/hr at 1 hr and greater represent a serious radiological hazard for a fairly long time after attack; sickness and possible fatalities among poorly sheltered or unsheltered people could result. For I_s values of 10,000 R/hr at 1 hr and greater (radiation levels that generally would occur only as a result of overlapping fallout patterns), survival would be possible only in the best available fallout shelters with facilities for an extended stay time plus decontamination requirements for a reasonably short reoccupation time after attack.

For the weather conditions usually assumed for the hypothetical counterforce and mixed attacks (the latter may include some ground bursts on urban targets) with a total delivered explosive yield of less than about 10,000 Mt, the areas within extremely high I_s contours would enclose less than 10% of the land area of the United States, and essentially all such areas would be rural forested or agricultural areas. Since the current Soviet nuclear force capability is estimated to be more than 10,000 Mt,[15] the delivery of such a force in a counterforce or mixed attack such as those represented in Figs. 1 and 2 would likely involve the coverage of more than 40% of the continental United States by I_s values of 100 R/hr at 1 hr and more than 25% of the area by I_s values of 1000 R/hr at 1 hr. Although these relative coverages of the land area are rather large, the associated degree of damage to or decrease in the yield of specific agricultural products by the respective exposures cannot be deduced from the curves of Figs. 1 and 2. To deduce damage, the relative geographic locations of the crops, targets, and assumed points of detonation, along with the meteorological inputs for each hypothetical attack must be considered; this procedure has been used in recent analyses.[16]

Future Weapon Systems

Over the past several years, one of the major developments in weapon systems has been the Multiple Independent Targeted Reentry Vehicle (MIRV). Certain information and estimates of apparent U.S.S.R. and U. S. progress and capabilities in the development of MIRV systems, especially with respect to their missile-carrying capacities, have been released to the press by various Department of Defense officials, including Secretary Laird. The following statements on Soviet nuclear force capabilities and MIRV system characteristics were provided by William Beecher[17] in a special article in the *New York Times,* Oct. 28, 1969:

> - As recently as last November, for example, the intelligence community predicted that the Soviet Union would stop deploying more intercontinental missiles when they had roughly equaled the 1054 in the American arsenal.

- The Soviet Union has in place or going into place about 1350 intercontinental ballistic missiles, roughly 300 more land-based units than the United States and 150 more than reported by American officials last spring.
- The Soviet Union has been testing a new swing-wing medium-range bomber, presumably for use against targets in Western Europe and Asia, even though it already has a fleet of 750 medium bombers. With aerial refueling, the new bomber could be used on round-trip strikes against the United States.
- The Russians are testing a new medium-range ballistic missile, though they already have more than 700 such missiles aimed at targets in Western Europe and Asia.
- The SS-9 can carry a single warhead of from 9 to 25 Mt (9 to 25 million tons of TNT) or three warheads of 4 to 5 Mt each. The SS-11 carries a warhead of 1 Mt, similar to the payload of the Minuteman missile.
- John S. Foster, Jr., the Pentagon's research and development chief, said that 420 SS-9's carrying three separately targetable warheads with one-quarter-mile accuracy could destroy about 95% of the 1000 Minutemen in their underground silos.
- The Soviet Union is now believed to have about 280 such giant missiles in various stages of construction. At the present rate of deployment, they could have the Minuteman killer force in three more years.
- The Minuteman-3 is designed to carry three warheads of about 100 kt, and the Poseidon submarine-based missiles, 10 warheads of 30 to 40 kt each. By comparison, the Soviet SS-9 is being tested with three warheads of about 5 Mt each, 50 times more powerful than each Minuteman-3 warhead.

On Jan. 6, 1970, the *Washington Daily News*, under a dateline from London, quoted the following statements:

- The Institute of Strategic Studies said the Soviet Union should have the capability to fit multiple nuclear warheads to its most powerful rockets by 1973.
- The influential study group, specializing in international defense developments, said the Soviets could have 500 of the multiple-warhead missiles ready for use by 1975.
- The multiple warheads are to be fitted to SS-9 Scarp rockets, "extremely powerful" three-stage missiles with a maximum range of 9800 miles, the report said. It estimated that each launcher cost between $25 and $30 million.
- About 250 of the SS-9's are believed to have been installed already in the Soviet Union, but these are not armed with the multiple warheads, the institute said.
- The Soviet SS-9 rocket originally was designed to carry a single warhead of between 10 and 25 Mt.

On January 7, 1970, Secretary of Defense Melvin Laird provided the following information to newsmen:

- The Russians could have a knockout missile force in place earlier than the 1974 period forecast to Congress last year.
- The discussion centered around Laird's estimate last summer that the Soviets could have about 420 of the huge SS-9 missiles in readiness by 1974. Such a force, Laird said then, could destroy 95% of this country's Minuteman missiles in a surprise first attack.
- He declined to say how many of the SS-9's, capable of hurling a single 25-Mt warhead or three warheads of 5 Mt each, are now in place or under construction. There have been unofficial estimates running up to about 279.

Defense officials, the news media, prominent scientists, and politicians have repeated similar information to the public over the past 10 months or more. The statements indicate that for the SS-9 missile the number of warheads apparently depends on the explosive yield of each warhead according to the relation

$$n_m = 866W^{-2/3} \qquad (1)$$

where n_m is the maximum number of warheads carried by the missile and W is the explosive yield of each warhead. Similarly, for the Minuteman-3 missile

$$n_m = 65W^{-2/3} \qquad (2)$$

and for the Poseidon and SS-11 missiles

$$n_m = 100W^{-2/3} \qquad (3)$$

Values of n_m for the SS-9 missile, the maximum explosive load, and the total target area enclosed by the 35-psi overpressure contour for selected warhead yields are given in Table 1 (for the case where all weapons are airburst at the height for which the area enclosed by the selected overpressure contour is

Table 1

CALCULATED VALUES FOR SS-9 MISSILE*

W $\left(\frac{\text{megatons}}{\text{warhead}}\right)$	n_m $\left(\frac{\text{warheads}}{\text{missile}}\right)$	n_mW $\left(\frac{\text{megatons}}{\text{missile}}\right)$	A_m (35 psi) $\left(\frac{\text{sq miles}}{\text{missile}}\right)$
0.1	40	4.0	31.8
0.3	19	5.7	31.4
1.0	8	8.0	29.5
3.0	4	12.0	30.7
10.0	1	10.0	17.1
25.0	1	25.0	31.9

maximized and at ground zero locations that are arranged in a hexagonal pattern in which the overpressure contours overlap in such a way that no point within the target receives less than 35 psi).

Table 1 shows that A_m (35 psi) is maximum at values of W which yield integer values of n_m in Eq. 1. For n_m equal to 2.0 warheads per missile, for example, W is 9.0 Mt. For this yield A_m (35 psi) is 31.9 sq miles/missile, although for the single 10-Mt warhead selected, A_m (35 psi) is only 17.1 sq miles/missile. In this case additional smaller warheads could be added to the capacity of the missile as appropriate to increase the value of A_m over that given. If the target area is less than 30 to 32 sq miles and n_m is more than 2, decoys could be used to replace some of the warheads.

Neglecting any possible effect of decoys and of active defense capabilities, the MIRV system using a maximum number of warheads, in contrast to a single warhead of maximum yield, apparently would provide no advantage in decreasing the number of missiles for imposing a selected minimum overpressure on a single target on an area basis. However, if the shape of the target area is considered, MIRV system weapons could achieve area enclosure within a selected overpressure contour with a smaller number of missiles and a smaller total explosive yield than could a single-warhead missile system. For example, a single SS-9 missile loaded with 40 100-kt warheads (4.0-Mt total yield) could, according to Table 1, enclose an area about 32 miles long and 1 mile wide within the 35-psi contour. Lengthwise coverage by the same overpressure contour would require five SS-9 missiles if each carried a single 25-Mt warhead (125-Mt total yield).

If the MIRV system could be employed with essentially no constraint on warhead dispersion among neighboring targets and if full use could be made of such capabilities to deliver warheads to targets, then any set of estimates of single-weapon missile force requirements may be directly converted to missile requirements for a system with MIRV. Under such conditions estimates of the number of SS-9 missiles required to cause specified levels of fatalities among the 1970 U. S. population sheltered in wood-frame structures exposed to selected minimum overpressures are given in Table 2 for weapon yields of 0.1, 1.0, and 10 Mt.* The lowest number of missiles for a given percentage of fatalities always occurs for a weapon yield of about 100 kt or less for the urban-center target areas.[20] Thus the general dependence of missile requirements on fatalities or area by a given overpressure contour relative to target size apparently ceases to be important for weapon yields less than about 100 kt. This independence is shown especially for the smaller high-density urban areas that would comprise the first set of targets for an antipopulation attack; a similar situation pertains for the smaller urban target areas listed in Table 2 in the range of 55 to 65% of the total population.

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*These estimates are based on information from the Japanese experience at Hiroshima and Nagasaki in World War II as discussed in Refs. 15, 18, and 19.

Table 2

ESTIMATED MINIMUM NUMBER OF SS-9 MISSILES WITH MIRV REQUIRED FOR SPECIFIED LEVELS OF FATALITIES AMONG THE 1970 U. S. POPULATION SHELTERED IN WOOD-FRAME STRUCTURES

	Minimum overpressure				
Fatalities, %	**5 psi**	**10 psi**	**15 psi**	**20 psi**	**35 psi**
			W = 0.1 Mt		
20	633	128	87	82	119
30	2,645*	528	220	184	250
40		5,770	470	348	473
50		21,820*	4,698	533	770
60			16,850	4,450	1,063
70			32,550*	12,210	5,310
80				51,630*	15,590
100					115,500*
			W = 1.0 Mt		
20	1,237	187	114	100	137
30	10,950*	1,094	270	223	292
40		6,615	903	420	542
50		24,020*	5,454	833	870
60			18,530	5,063	1,179
70			35,430*	13,520	5,832
80				55,930*	16,920
100					124,500*
			W = 10 Mt		
20	6,848	924	427	340	324
30	23,570*	1,541	1,408	707	669
40		16,260	6,178	1,707	1,173
50		46,000*	14,010	5,008	2,208
60			36,520	13,290	4,319
70			65,610*	26,910	14,270
80				100,900*	33,360
100					218,800*

*F_f(max) is 0.28 at 5 psi, 0.45 at 10 psi, 0.62 at 15 psi, 0.80 at 20 psi, and 1.00 at 35 psi.

In other words, a further significant reduction in overkill and wastage of explosive energy associated with the detonation of large-yield weapons on small-size targets would not be achieved by the use of weapons with yields less than 100 kt on U. S. urban centers as a target system. However, for attacks designed to cause more than about 65% fatalities under the conditions assumed in Table 2, the various states or the country as a whole would become a single target, and on an area basis the number of missiles required would be essentially independent of weapon yield for missiles carrying maximum payload.

The Soviet's estimated 1970 intercontinental nuclear force, from the previously quoted statements, is approximately 11,000 Mt, assuming a one-way mission or refueling of 750 bombers carrying a payload of 5 Mt each, 1100 SS-11's carrying 1 Mt each, and 250 SS-9's carrying 25 Mt each. These estimates do not include the submarine force of perhaps 200 vessels, because it is assumed that its mission would be that of a reserve or second-strike force. Such a ready force, if delivered in an antipopulation attack with 100% reliability and accuracy in the most efficient manner (i.e., by allocating the 1-Mt weapons to densely populated cities with small areas and the 25-Mt weapons to less densely populated urban centers covering larger areas) utilizing full-target coverage by 20- or 35-psi overpressure contours, could result in fatalities amounting to about 42% of the population if all were sheltered in wood-frame structures. This percentage of fatalities is equivalent to the entire 1970 population of the 680 largest U. S. cities.

If this same nuclear striking force were converted to efficient and maneuverable MIRV systems with 100-kt warheads, the single 5-Mt warhead assumed for the bombers would convert to thirteen 100-kt warheads; the single 1-Mt warhead taken for the SS-11 would convert to four 100-kt warheads; and the single 25-Mt warhead for the SS-9 would convert to about forty 100-kt warheads. The combined striking power for these warheads is then 2375 Mt, which, if delivered according to the assumptions in Table 2, could produce about 52% fatalities among the 1970 U. S. population. This combined nuclear striking force would be equivalent to a total of 593 deployed SS-9 missiles with the MIRV system, all armed with 100-kt warheads.

Assuming such an SS-9 MIRV force to be in existence, estimated minimum deployment times and costs in 1970 U. S. dollars for both the total and additional SS-9 missiles (each fitted with 40 100-kt warheads) required to cause stated relative fatality levels among the 1970 U. S. population for the conditions of Table 2 are given in Table 3. The year of final deployment is based on the assumption of both a constant rate of production and one that increases linearly from 50 to 100 missiles per year from 1970 to 1980.

Note that the calculations are based on the 1970 U. S. population distribution; thus, for the fatality percentage having Y_1 and Y_2 values significantly larger than 1970, the number of required missiles, the values of Y_1 and Y_2, and the added cost are all underestimates (except for the 100% level of fatalities). Since the estimated number of missiles refers to weapons delivered on target, these figures are, by definition, underestimates of force requirements for the stated fatality levels.

These estimates suggest that at the current rate of production the most economical and effective SS-9 MIRV system could not impose, through air-blast weapons effects, the current popular view of assured and complete destruction of the 1970 U. S. population in a nuclear attack until sometime after the year

Table 3

ESTIMATED NUMBER OF SS-9 MISSILES WITH MIRV, YEAR OF FINAL DEPLOYMENT, AND COSTS FOR CAUSING A STATED PERCENTAGE OF FATALITIES AMONG THE 1970 U. S. POPULATION BY BLAST EFFECTS ON PEOPLE IN WOOD-FRAME STRUCTURES

Fatalities, %	Total required number of SS-9 missiles	Additional required number of SS-9 missiles	Y_1^* (year)	$Y_2^\dagger$ (year)	Added cost, 10^9 \$ (U. S., 1970)
20	80				
30	180				
40	340				
50	530				
60	1,060	467	1979	1977	13
70	5,310	4,717	2064	2005	130
80	15,600	15,010	2270	2037	413
90	38,700	38,110	2732	2084	1050
100	115,500	114,900	4268	2175	3200

*$Y_1 = 0.02N_m + 1958$, at a constant rate of 50 SS-9 missiles per year.

†$Y_2 = (0.4N_m - 137)^{1/2} + 1960$, at a rate of 50 (1 + 0.1t) SS-9 missiles per year, where $t = Y_2 - 1970$.

3000. With a constantly increasing rate of production of the missile system, however, the force required for such a level of fatalities might be assembled and deployed in 100 to 200 years. The cost of such a system could be two times the estimated \$3 trillion (1970 dollars); this is about 1000 times the current yearly Gross National Product of the U.S.S.R.

Methods for estimating the intermediate-range fallout from 100-kt-yield weapons detonated as airbursts to give maximum area coverage of a given overpressure contour are not immediately available. Thus the general extent or degree of the radiological hazard to agricultural areas downwind from any of the larger urban centers hit in such an attack cannot be given.

The effects of detonating the 100-kt weapons at ground level were investigated in an alternate assumed attack mode. This alternative is suggested since the fallout levels in the vicinity of ground zero appear to be maximized at a yield of around 100 kt. The areas enclosed by exposure-dose contours of 400 and 1200 R over a period of 100 hr after fallout arrival for 100-kt-yield (100% fission) and 1-Mt-yield (50% fission) surface detonations are shown in Fig. 3. The 400-R contour indicates generally the limiting extent (outer boundary) at which a significant number of persons sheltered in wood-frame houses would experience radiation sickness. The 1200-R contour indicates generally the limiting boundary at which essentially all persons sheltered in wood-frame houses over the specified 100-hr period would eventually die. In other words, all

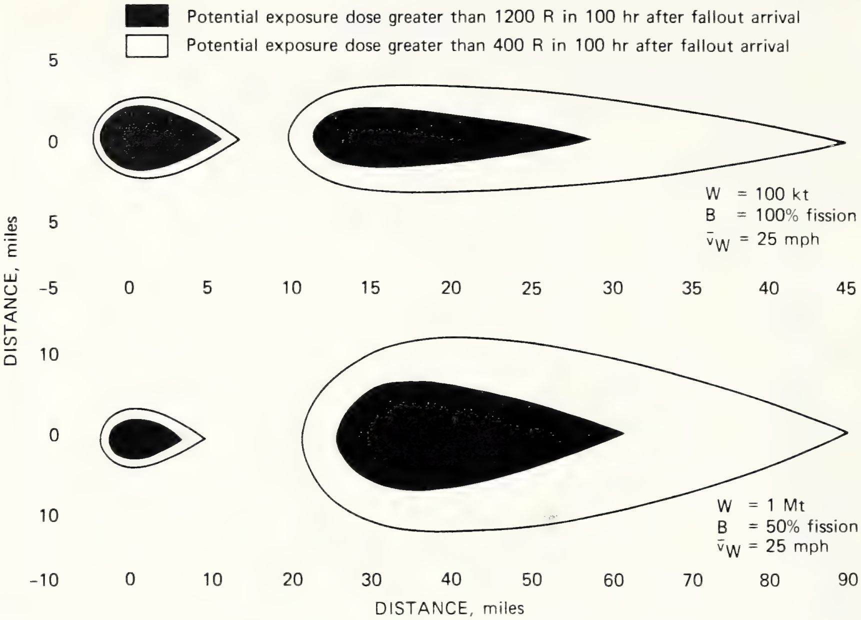

Fig. 3 Area enclosed by exposure-dose contours of 400 and 1200 R for fallout from 100-kt- and 1-Mt-yield surface detonations.

persons in the shaded area of Fig. 3 who were in shelters with a protection factor of 2 (or more at central locations) would become fatalities.

The full significance of the total fallout-radiation hazard within the two elliptically shaped areas, in terms of number of fatalities, cannot be readily incorporated into the described antipopulation attack patterns (in which the only hazard considered was blast overpressure from air detonations) without using a large-scale computer program. However, a conceptional view of the relative hazard to people in small wood-frame structures can be obtained from simple arithmetic estimates if only the circular portion of the potentially lethal area around ground zero is considered. The radius of this area is 1.9 miles for the 100-kt detonation and 2.5 miles for the 1-Mt detonation. Thus the potential lethal radius for the fallout hazard from the 100-kt surface detonation under the assumed exposure conditions is 3.4 times the lethal radius for the overpressure hazard from the 100-kt air burst (the area ratio is almost 12 to 1). In comparison, these radius and area ratios for the 1-Mt detonation are only 2.1 and 4.4, respectively. Another way of stating the relative extent of these two hazards for the specified exposure conditions is that the area coverage of the 100% lethal fallout level from a 100-kt surface burst is equal to that of the overpressure effects from a 4-Mt air detonation.

For people inside concrete buildings with a protection factor of 100, the radius of the 600-R lethal-exposure dose from ground-zero-region fallout from the 100-kt surface detonation is almost 0.5 mile, only slightly larger than the radius of the 48-psi contour (100% lethal for occupants of concrete structures) for the 100-kt air burst. The same relative potential hazard from fallout does not occur for the 1-Mt surface detonation since the absolute magnitudes of the fallout levels near ground zero are smaller in this case; also, the time of fallout arrival is shorter for the smaller-yield detonation.

The general dispersion of ground-zero- and downwind-fallout patterns, as represented by the 1200-R potential-exposure-dose contour, for closepacking of the ground-zero patterns to cover circular-shaped urban areas is illustrated in Figs. 4 and 5. In these figures A_T is the largest inscribed circular area enclosing an urban target area, and A_R is the area within the downwind 1200-R exposure-dose perimeter for fallout from cloud altitudes. Figure 5 shows that A_T for 16 and 28 detonations includes a portion of several cloud-fallout patterns; in addition, the maximum downwind extent of the perimeter of the

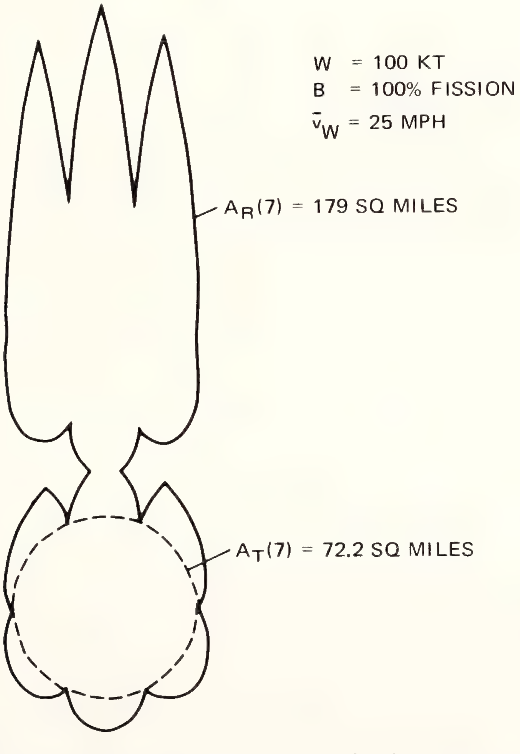

Fig. 4 Geometric configuration of A_T and A_R for the 1200-R exposure-dose perimeter when A_T is equal to the maximum circular area covered by seven overlapping ground-zero fallout patterns.

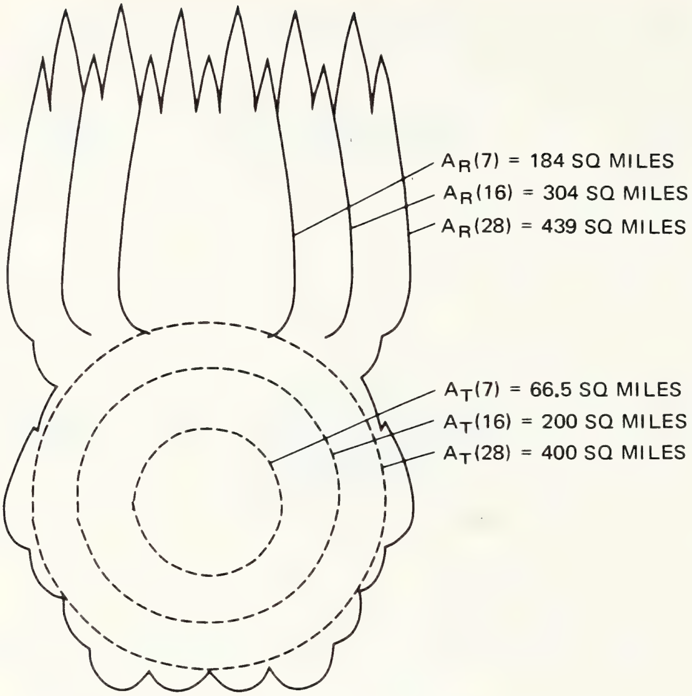

Fig. 5 Geometric configuration of A_T and A_R for the 1200-R exposure-dose perimeter when A_T is equal to the maximum circular area covered by 7, 16, and 28 overlapping ground-zero fallout patterns.

1200-R exposure-dose contours is essentially constant and independent of the size of the circular target.

The average, or midrange, values of A_T and A_R are plotted as a function of the number of weapons detonated (or the number of ground-zero patterns) giving full circular coverage of the target area. No real, single, smooth curve of A_T and/or A_R as a function of the number of detonations or weapons exists for target-area coverage requiring one to seven weapons per target. The curves in Fig. 6 tend to follow midrange values of A_T and A_R; as the number of weapons per target increases, the percentage spread in possible values of these two parameters decreases. The curves in Fig. 6 were used to estimate the number of weapons per target required to enclose each of the 500 largest U. S. cities or urban places within the 1200-R contour and the relative amount of land area outside the urban areas that would also be enclosed (assuming no overlapping of the fallout patterns from these targets and no loss of fallout to areas outside the country). The calculated cumulative explosive yield of the 100-kt weapons, the total rural land area enclosed, and the number of people involved (i.e., those

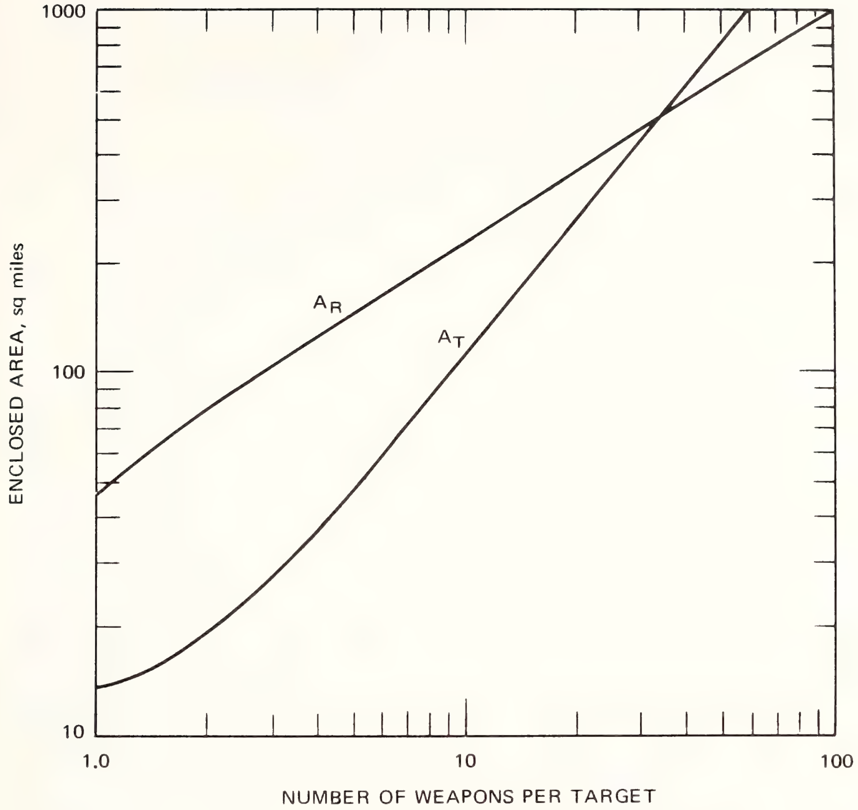

Fig. 6 Variation of A_T and A_R with number of weapons detonated or number of ground-zero fallout patterns.

residing in the area given by A_T who would be fatally involved if sheltered in wood-frame houses) are given in Table 4.

The calculations in Table 4 indicate that, for the 500 most densely populated cities or urban places, coverage of the respective A_T with at least the 1200-R exposure-dose contour could be accomplished with a total explosive yield of about 103 Mt (i.e., 1030 delivered weapons yielding 100 kt each) and that almost 1.0% of the land area of the United States outside the cities would be enclosed within the specified 1200-R exposure-dose contour. As shown, the 500 most densely populated cities or urban places contain about 35% of the 1970 U. S. population; the estimated number of 100-kt airbursts required to cause 35% fatalities by air-blast effects among the population sheltered in wood-frame structures would be about 14,000.

Thus, if the major portion of the U. S. population were in shelters with a protection factor of 2 at the time of attack, the number of SS-9 missiles with an

Table 4

CUMULATED TOTAL YIELD OF 100-KT SURFACE BURSTS TO ENCLOSE THE 50 TO 500 MOST DENSELY POPULATED CITIES AND NEARBY RURAL AREAS WITHIN THE 1200-R EXPOSURE-DOSE CONTOUR

Target number	M, Mt	A_R, sq miles	$A_R/3.6 \times 10^4$, %	Cumulated percent of total population
50	15.2	4,470	0.12	12.8
100	23.3	7,520	0.21	15.9
200	41.5	14,190	0.39	21.4
300	62.3	21,240	0.59	27.1
400	79.0	27,690	0.76	30.4
500	103.4	35,750	0.99	35.0

idealized MIRV system and 100-kt weapons required to cause a given level of fatalities would be about a factor of 13.5 less for an attack in which all explosions are ground bursts instead of airbursts (i.e., if the fallout effect instead of the air-blast effect were used against the population). This result suggests that, without reasonably good fallout protection in the cities, the planned use of surface-detonated 100-kt weapons could reduce the time scale required to construct a force that could assuredly destroy the 1970 U. S. population from about a century or two to about a decade or two (especially if all technical problems of production of such a force would be solved without causing extended delays in deployment).

This rather high relative degree of potential effectiveness of the fallout hazard from the 100-kt surface detonation could, of course, be countered by the provision and use of shelters with protection factors higher than 2. Increasing the protection against the fallout radiation would decrease the lethal radius from fallout radiation. In turn, a larger number of warheads and missiles would be required to accomplish the same level of population destruction by either fallout radiation or air blast. The times for producing the needed force would then be increased beyond the minimum of the decade or two indicated previously. For a shelter protection factor of 130, the 100% lethal radius for the very close-in fallout from a 100-kt true surface burst would be equal to the 100% lethal radius for the population sheltered in concrete buildings subjected to the air-blast overpressure from a 100-kt airburst. In such a protective posture, the limiting force requirements for assured destruction of the 1970 population would be 160,000 SS-9 missiles carrying 100-kt warheads with MIRV's for the smaller targets. Such a force, built at the previously assumed rates which increase continuously with time, could be deployed approximately by the year 2760 at a cost of about $4.5 trillion (1970 U. S. dollars).

The apparent advantage of the reduction in force requirements gained when lower-yield warheads with MIRV are allocated to small urban places, missile sites, and other military targets, together with the fact that area coverage for the circular prompt weapons-effect contours is independent of weapon yield, could suggest a gradual conversion of existing stockpiled weapons to lower-yield warheads in all nuclear arsenals soon after MIRV capabilities become operational. If this is the case, some major changes in civil-defense policies, programs, and operational plans could be considered to provide an appropriate response to salient features of the revised-force capabilities. Two major options are: (1) the provision of increased protection to the population and to other resources in urban areas against the prompt weapons effects (i.e., blast, thermal, shock, and initial nuclear radiation) and (2) the evacuation of cities when there is sufficient warning time. The first option would include the provision of shelters with a minimum protection factor of 130 to negate the advantage of the 100-kt surface burst. For the second option, some difficulties could occur if ground bursts were used; however, the downwind extent and width of the fallout pattern from the 100-kt surface detonation is much less than that from detonations in the megaton-yield range, as shown in Fig. 3. This associated reduction in fallout areas for attack patterns including only urban-area targets (65% or less of the 1970 population) would leave essentially all the rural areas and the agricultural sector free of direct exposure to any weapons effects. If shelters were available in urban areas, postattack evacuation to rural areas free of fallout would be a feasible operational alternative.

As previously mentioned, exposure doses from fallout radiation near ground zero are greater for detonations with yields close to 100 kt because of the early fallout arrival times and the rather heavy local deposits surrounding the point of detonation. Further insight into these ramifications of the fallout hazard would require a more detailed analysis than that given here; such an analysis could be readily accomplished with the aid of computers. Specific consideration of the people, animals, and plants that could be exposed to radiological hazards from the downwind fallout has been neglected here. However, practically no human fatalities would occur from fallout in the downwind area from the 100-kt surface burst if shelters with a protection factor of 130 were available and were used. In the described antipopulation attacks (similar results would apply to a pure counterforce attack), the downwind boundary of the 1200-R exposure-dose contour extends a distance of 20 to 30 miles from the downwind edge of the urban areas. Thus the size of the rural farm areas receiving moderately heavy fallout levels from the 100-kt surface bursts would be approximately equal to the size of the urban areas subjected to direct attacks. Consequently agricultural problems caused by fallout would be limited to regions near target cities or target military installations. This pattern would persist until more than about 65% of the population (all urban places) was involved. For much heavier attacks, with 100-kt-yield ground-burst weapons, however, the radiological effects on agriculture could approach those predicted for the counterforce and mixed attacks using larger-yield weapons.

SUMMARY AND CONCLUSIONS

The intrinsic properties of fallout associated with radiological hazards which could affect agricultural operations in the postattack period of a nuclear war include: (1) the radionuclide composition of the fallout material, which determines the energy composition of the gamma and beta radiation emitted, (2) the physical and chemical properties of the fallout particles (such as size, shape, composition, structure, and solubility) which influence their retention by surfaces, and (3) the solubility and biological availability of specific radionuclides. In terms of crop or agricultural-product output, both operational factors (effects on man and his social system) and biological factors (response of plants, animals, birds, and insects) would be important.

The degree of the hazard to the food-producing agricultural systems would generally depend more on external parameters, such as the available weapon system, form or mode of attack, level of attack, explosive yield of weapons, relative heights of burst, and local and regional weather patterns, than on the properties of the fallout. The latter would tend to influence the form rather than the degree of the hazard.

A major recent development in weapon systems that could have a significant impact on the type and extent of hazard to agriculture in a nuclear war is the Multiple Independent Targeted Reentry Vehicle. Indeed, estimates of MIRV system capabilities, especially in terms of using many smaller-yield warheads on many smaller targets, may be used to identify several important implications for future civil-defense planning. One estimate involves the relatively high levels of the fallout hazard near ground zero, which apparently has a maximum for a surface detonation at a yield of about 100 kt. The implication of this effect on weapon-system cost and times of deployment for the Soviet Union and its SS-9 missile system is that, if the U. S. fallout-shelter system were poor and a majority of people had to remain in their houses during an attack, the Soviets could build and deploy at a cost of about $30 billion within the next 10 to 20 years a nuclear force of sufficient capability to essentially assure the destruction of the entire U. S. population. In this case "sufficient capability" refers to the use of the force in an antipopulation attack in which local fallout would be the main cause of fatalities. On the other hand, if fallout shelters with protection factors of 130 or more were available and were used, no advantage in force requirements would accrue by the use of surface bursts. Instead, the more reliable overpressure effects would be used. In the limit, the assured destruction of the U. S. population by blast effects would require at least 160,000 SS-9 missiles. Even at reasonable increases in production rates, the Soviets would have difficulty in deploying such a force within the next 800 years (using currently available technologies); the cost of such a force would be prohibitive at more than $4.5 trillion (1970 U. S. dollars).

The major implication for agricultural systems of the possible use of MIRV and the associated lower-yield warheads in a nuclear war is that the fallout would be of the intermediate or worldwide type for attacks in which air-blast effects are emphasized and that, where the fallout effects are emphasized by use of ground bursts, the heavy downwind deposits of local fallout would be limited to a distance of about 30 miles from the downwind edge of any target independent of the size of the target. In other words, the effect of MIRV and the associated lower-yield warheads would be to almost eliminate the widespread fallout effects previously estimated for attacks in which land-surface detonations of weapons in the megaton-yield range have been postulated. With the described Soviet SS-9 missile system with MIRV capabilities, a comparable degree of effect on agriculture might be achieved from attacks designed to kill more people than the entire U. S. urban population (i.e., more than 65% of the 1970 U. S. population) in which all detonations programmed for the rural areas would be surface bursts. Further detailed calculations are required before the potential of such an attack to cause significant adverse effects on agriculture can be evaluated, given the current public fallout-shelter system as a basis for estimating population survival.

REFERENCES

1. C. F. Miller and P. D. LaRiviere, Introduction to Longer-Term Biological Effects of Nuclear War, SRI Project IMU-5779, Stanford Research Institute, 1966.
2. R. B. Platt, Ecological Effects of Ionizing Radiation on Organisms, Communities, and Ecosystems, in *Radioecology,* Reinhold Publishing Corporation, New York, 1963.
3. C. F. Miller, Fallout and Radiological Countermeasures, Vols. I and II, SRI Project IMU-4021, Stanford Research Institute, 1963.
4. K. H. Larson and J. W. Neel, Summary Statement of Findings Related to the Distribution, Characteristics, and Biological Availability of Fallout Debris Originating from Testing Programs at the Nevada Test Site, USAEC Report UCLA-438, University of California, Los Angeles, Sept. 14, 1960.
5. J. P. Friend, H. W. Feely, P. W. Krey, J. Spar, and A. Walton, The High Altitude Sampling Program, Report DASA-1300 (Vols. 1, 2A, 2B, 3, 4, and 5), Defense Atomic Support Agency, Aug. 31, 1961.
6. C. F. Miller, Operation Ceniza-Arena: The Retention of Fallout Particles from Volcán Irazú (Costa Rica) by Plants and People, Parts Two and Three, SRI Project IMU-4890, Stanford Research Institute, 1967.
7. National Academy of Sciences—National Research Council, Damage to Livestock from Radioactive Fallout in Event of Nuclear War, Publication No. 1078, Washington, D. C., 1963.
8. National Committee on Radiation Protection and Measurements, Exposure to Radiation in an Emergency, Report No. 29, 1962.
9. J. L. Mackin, S. L. Brown, and W. B. Lane, Beta Radiation Dosimetry for Fallout Exposure Estimates: Comparison of Theory and Experiment, SRI Project 7402, Stanford Research Institute, 1969.

10. S. L. Brown, Disintegration Rate Multipliers in Beta-Emitter Dose Calculations, SRI Project IMU-5116, Stanford Research Institute, 1965.
11. J. D. Teresi and C. L. Newcombe, An Estimate of the Effects of Fallout Beta Radiation on Insects and Associated Invertebrates, Report AD-633024, Naval Radiological Defense Laboratory, Feb. 28, 1966.
12. Report of ICRP Committee on Permissible Dose for Internal Radiation (1959), *J. Health Phys.*, 3 (1960).
13. J. L. Kulp and A. R. Schulert, Strontium-90 in Man and His Environment, USAEC Report NYO-9934 (Vols. I, II, and III), Columbia University, 1961 and 1962; also R. S. Hirshman and A. R. Schulert, Strontium-90 Assay of Various Environmental Materials, USAEC Report NYO-9934(Vol. III) Ext., Columbia University, 1961.
14. C. F. Miller and S. L. Brown, Models for Estimating the Absorbed Dose from Assimilation of Radionuclides in Body Organs of Humans, SRI Project IMU-4021, Stanford Research Institute, 1963.
15. C. F. Miller, Assessment of Nuclear Weapon Requirements for Assured Destruction, URSRC Report 757-6, URS Research Company, 1970.
16. R. K. Laurino, National Entity Survival Following Nuclear Attack, SRI Project IN-OAP-28, Stanford Research Institute, 1967.
17. William Beecher, Soviet Arms Gain Detected by U. S., *New York Times,* 1969.
18. Ashley W. Oughterson and Shields Warren, *Medical Effects of the Atomic Bomb in Japan,* Division VIII, Vol. 8, National Nuclear Energy Series, McGraw-Hill Book Company, Inc., New York, 1956.
19. Raisuke Sirake, Medical Survey of Atomic Bomb Casualties, Research on the Effects and Influences of the Nuclear Bomb Test Explosives, Volume II, Japan Society for the Promotion of Science, Tokyo, 1956.
20. C. F. Miller, Protection of People by Structures from the Initial Radiation, Blast, and Thermal Phenomena of Nuclear Explosives, Research Report No. 7, Office of Civil Defense, Department of Defense, 1962.

RETENTION OF SIMULATED FALLOUT BY SHEEP AND CATTLE

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ABSTRACT

The initial retention of 88- to 175-μ or 175- to 350-μ near-in fallout-simulant sand on the backs of cattle averaged 50%. This was independent of mass loading up to 100 g/m^2. The retention half-time of simulant deposited on the animals' backs averaged 9 days for cattle kept under feedlot conditions and 2 days for cattle kept under pasture conditions.

The fecal excretion of simulant sand given to sheep and cattle could be described by single exponential functions. The mean lifetime ($1.44T_{1/2}$) of material in the gut averaged 1.1 days in sheep and 4.8 days in cattle.

The beta-particle radiation dose to grazing and feedlot animals from near-in fallout would be principally due to retention of particulates on the body and their passage through the gut.

RETENTION ON THE SURFACE OF ANIMALS

Near-in fallout-simulant sand was spread as an aerosol over 26 Hereford and Angus cattle by means of a blower. The sand, which was labeled with ^{177}Lu for identification by gamma-ray spectrometry, was in the particle range either 88 to 175 μ or 175 to 350 μ. The aerosol was generated at a height sufficient to guarantee terminal velocity before deposition. Initial retention was determined by comparison with deposition on disk impactors. A 0.6-cm-thick 7.5-cm-diameter NaI(Tl) scintillation crystal was used for counting.

The initial retention, as well as the retention as a function of time, was dependent on the location on the surface of the animal's back. The mean initial retention of all the sites monitored was about 50% for studies using the 88- to 175-μ sand. For the 175- to 350-μ size the mean initial retention was also near 50%.

Figure 1 shows the retention vs. time at four different locations for one cow. Loss is reasonably rapid for the convex locations and is minimal for the flat or concave locations.

Retention was also strongly dependent on the activity of the animals. Figure 2 compares retention on animals kept under feedlot conditions with that on animals allowed the greater mobility of pasture conditions. The retention half-time for pastured animals was less than 2 days but was greater than 9 days for animals kept under feedlot conditions. The loss rate for the coarse sand was slightly greater than for the fine sand.

In summary, the greatest beta-radiation skin dose would be to the region between the hook bones, and, since most U. S. cattle are kept under pasture conditions, a retention half-time of 2 days should probably be used in the dose calculation.

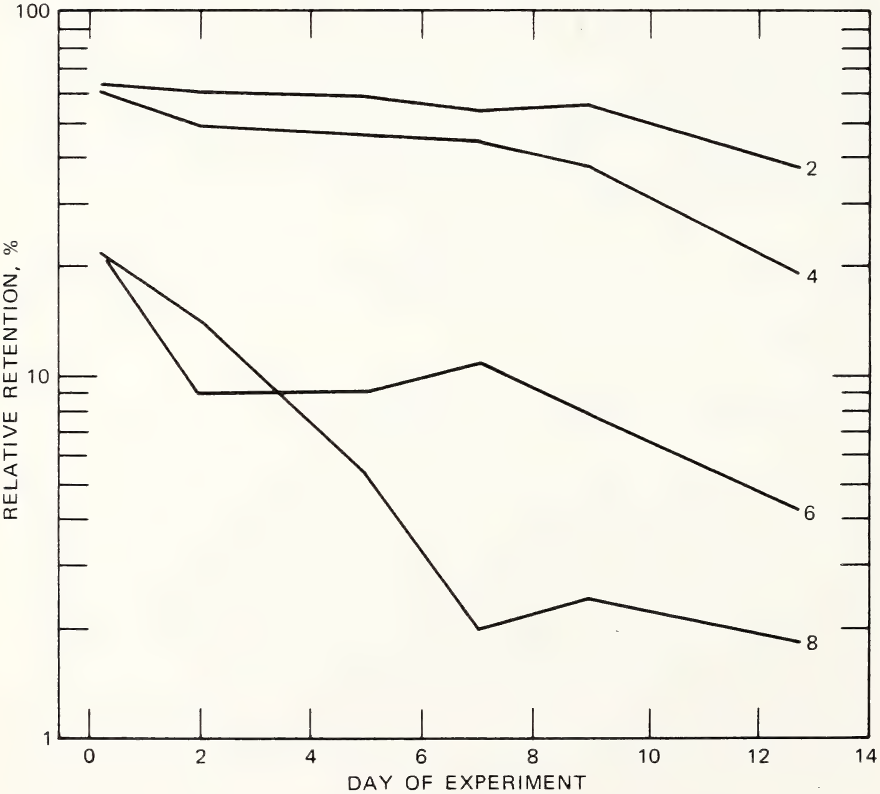

Fig. 1 Typical retention of 88- to 175-μ sand at four locations on the back of cow 712: curve 2, between tuber coxa; curve 4, between shoulders; curve 6, paralumbar fossa; and curve 8, over shoulder joint.

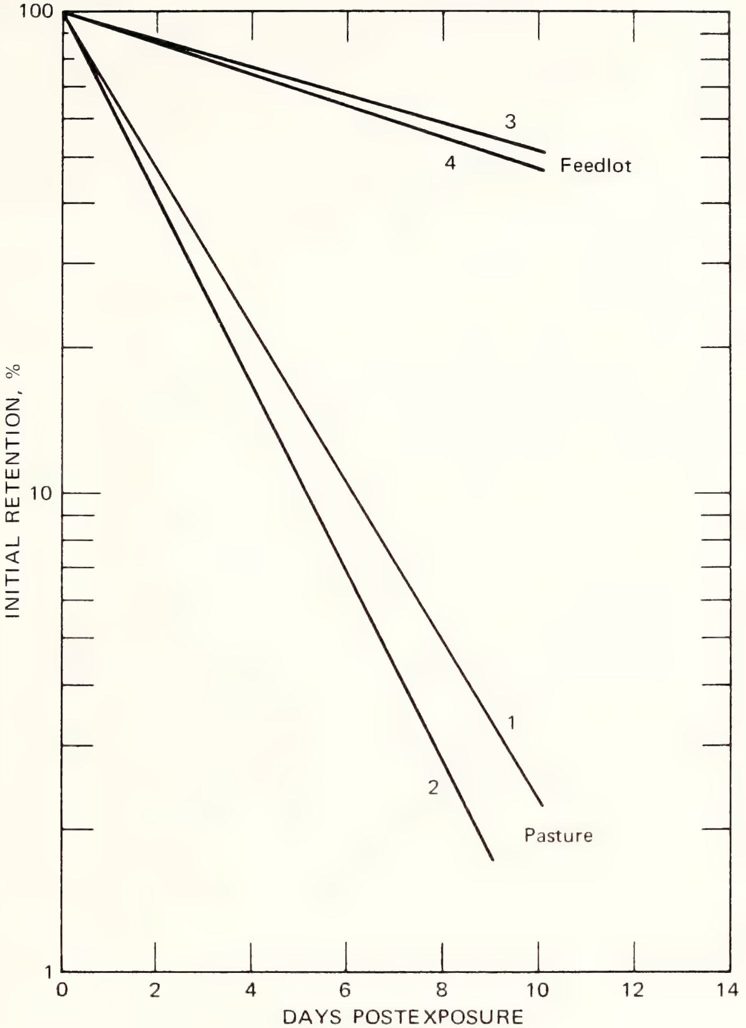

Fig. 2 Composite normalized retention on the backs of cows, comparing particle size and cattle kept under pasture conditions and under feedlot conditions. Curves 1 and 3 are for 88- to 175-μ sand; curves 2 and 4, for 175- to 350-μ sand.

RATE OF PASSAGE OF NEAR-IN FALLOUT IN THE GUT

The radiation dose to segments of the gut would be from unabsorbed near-in fallout reaching the gut by either ingestion or inhalation. The radiation dose to the whole gut or to any segment is proportional to the average time that particles spend in any location. The definition of the mean retention time or mean transit time is the summation of times that individual particles spend in the gastrointestinal tract divided by the total number of particles. By this definition, however, mean retention time is very difficult to determine.

If the ruminant gut is considered in a one-compartment model where mixing of digesta is very rapid and emptying is by first-order kinetics, the mean retention time can be calculated very simply. It is the reciprocal of the first-order rate constant, or 1.44 times the biological half-time. A typical excretion curve of 88- to 175-μ sand particles in sheep is given in Fig. 3.

The true estimate of the mean retention time (τ) from such data is the weighted average abscissal value (i.e., the centroid of the curve), and, if the function of the curve is not known, mean retention time must be determined by approximation methods. From our data, however, the area under the buildup portion of the curve is small compared with the total area, and there is little error in calculating τ from the measured half-life.

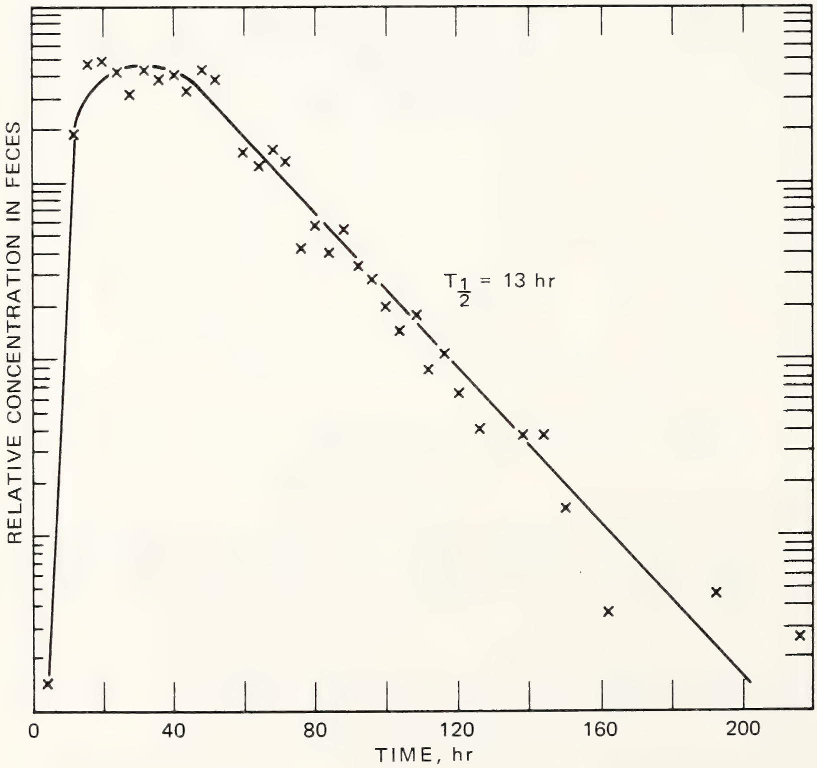

Fig. 3 Typical excretion function of a single dose of 88- to 175-μ ^{177}Lu-labeled sand in sheep.

There is actually a great amount of data in the animal-science literature on the rate of passage of digesta in ruminants since this is an important factor in determining nutritional efficiency of feedstuffs.[1] Rate of passage as calculated from our model is simply the average mass of rumen digesta at any time divided by τ.

The values observed from our study are shown in Table 1. They are, in general, greater than those reported in the literature for feedstuffs of the same particle size. The data in Table 1 show little difference caused by sand particle size but an appreciable difference between sheep and cattle.

Table 1

MEAN LIFETIMES OF SIMULATED NEAR-IN FALLOUT IN THE GUT OF SHEEP AND CATTLE

Sand size, μ	Lifetime in sheep, days	Lifetime in cattle, days
88 to 175	1.2	4.8
175 to 350	1.1	

A very important finding was that for both sizes of sand and with both sheep and cattle there was 98 to 100% recovery. This implies that very little, if any, of the sand particles are trapped in fine structures of the GI tract.

Again we must stress that our data correspond to normal intake conditions. If dose to the GI tract is sufficient to cause appreciable damage, then decreases in motility are to be expected; this would increase retention times and further increase the dose.

REFERENCE

1. C. C. Balch and R. C. Campling, Rate of Passage of Digesta Through the Ruminant Digestive Tract, in *Physiology of Digestion in the Ruminant,* pp. 108–123, Second International Symposium, Ames, Iowa, 1964, R. W. Dougherty et al. (Eds.), Butterworth & Co. (Publishers) Ltd., London, 1965.

SIMULATED-FALLOUT-RADIATION EFFECTS ON SHEEP

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ABSTRACT

Sixty-four yearling lambs were exposed to the following radiation treatments: (1) ^{90}Y beta irradiation of the gastrointestinal tract (2.4 mCi/kg of body weight for 3 consecutive days, (2) ^{90}Y beta irradiation of the skin (57,000 rads), (3) ^{60}Co irradiation of the total body (240 R), or (4) all possible combinations of these treatments. Irradiation of the gastrointestinal tract produced severe injury to the rumen and abomasum and resulted in severe anorexia and diarrhea and a significant loss ($>20\%$) of body weight. Nearly 50% of the lambs subjected to combined gastrointestinal and whole-body irradiation died within 60 days, but lambs in other treatment groups were able to recover from the initial irradiation insult. Skin irradiation caused no immediate threat to life but affected survival several months postirradiation. Implications of multiple irradiation trauma on animal survival are discussed from a postattack recovery viewpoint.

In the event of a surface thermonuclear detonation, farm livestock located downwind from the site of attack would be vulnerable to fallout radiation. The response of grazing livestock to fallout radiation would result from the combined insults of external whole-body gamma irradiation, irradiation from contaminated feed, and beta irradiation to animals' skin. Considerable information is available on the effects of whole-body gamma radiation on large animals,[1,2] and incidences of skin irradiation from radioactive fallout have been reported in livestock[3,4] as well as in man.[5] Less is known about the response of the gastrointestinal tract of large animals to ingested radioactive materials. Nold, Hayes, and Comar,[6] measuring internal radiation doses in dogs and goats using implanted glass-rod dosimeters, reported that, when a soluble ^{90}Y solution was given, the greatest doses were measured in the lower large intestine. Lethal levels of ingested soluble ^{144}Ce–^{144}Pr severely damaged the rumen and omasum of sheep.[7] More recently it has been shown that ingestion by sheep of insoluble ^{90}Y-labeled fallout simulant at levels to be expected in fallout contamination severely affected animal health and productivity but was seldom lethal.[8] Furthermore, the sites of major damage were confined to the rumen and abomasum.

Even though previous studies demonstrate the effects of radiation on livestock, few studies have been conducted to determine the interaction of simultaneous administration of multiple modes of irradiation. Baxter et al.[9] reported that the additional trauma of thermal burns increased mortality in whole-body X-irradiated (400 R) swine. George, Hackett, and Bustad[10] irradiated lambs by three different methods (whole-body X-ray, oral ^{131}I, and beta irradiation of the skin) to study the additive effects at two planes of nutrition. None of the single or combined treatments were lethal, and weight gain appeared to have been influenced mainly by the nutritional treatments. The need for information on the survival of large animals in a postattack fallout situation was recently emphasized,[11] and this study was initiated to investigate these interactions.

EXPERIMENTAL PROCEDURES

Yearling wether lambs of mixed breeding were treated for parasites, shorn, and gradually adjusted to a 680-g ration of pelleted alfalfa preconditioned with 140 g of water. The ration, which was supplemented with trace-mineralized salt, represented about 80% of ad libitum consumption. The sheep, averaging 31.1 ± 0.6 kg in weight, were placed in collection stalls approximately 7 days before irradiation. One wether was randomly assigned to each of eight treatment groups: (1) control, (2) gastrointestinal irradiation (GI), (3) whole-body gamma irradiation (WB), (4) skin irradiation (Skin), (5) WB + Skin, (6) GI + Skin, (7) GI + WB, and (8) GI + WB + Skin. Eight replicates of each treatment were made over a period of 9 months.

A sublethal bilateral exposure of 240 R (midline dose of 145 rads) at 1 R/min from ^{60}Co sources[12] was used for whole-body gamma irradiation. Sheep assigned to the four treatments requiring gamma irradiation were simultaneously irradiated 12 to 16 hr before gastrointestinal and skin irradiation began. Four 43-by-28-cm, flexible, sealed ^{90}Sr—^{90}Y plaques[13] with surface dose rates ranging from 913 to 1570 rads/hr were used to irradiate about 12% of the body area. A plaque was affixed to the thoracolumbar region of the back of each sheep and left until a total beta dose of 57,000 rads had been delivered. The ratio of skin beta dose to whole-body gamma dose in the combined treatments was 240 to 1—the ratio estimated for the cattle exposed during the Trinity shot[3] in 1945.

The insoluble labeled fallout simulant (^{90}Y-labeled silica sand 88 to 175 μ in size) was mixed with the daily ration and fed for 3 consecutive days, as previously described.[8] An initial activity of 2.4 mCi/kg of body weight was fed on day 1, but, because of ^{90}Y decay, only 1.8 and 1.4 mCi/kg remained when the ration was fed on days 2 and 3, respectively. The specific activity of the various batches of sand ranged from about 5 to 10 mCi/g; thus 6 to 17 g of sand were fed daily.

The half-life, energy, and particle size of the synthetic fallout were selected to simulate fallout from a 1-Mt or greater surface nuclear burst at a distance sufficient for most livestock to survive the gamma dose. The gastrointestinal dosimetry procedure and results are described in detail by Wade et al.[14]

Consumption of feed and water and excretion of feces and urine were recorded daily. Six to seven weeks after treatment, the animals were removed from the collection stalls and fed an alfalfa–grass hay and grain ration ad libitum. Body weights were recorded periodically throughout the study.

Fecal samples were oven dried at 60°C, and bremsstrahlung was counted with a well-type gamma scintillation counter set to exclude all pulses less than 2 MeV. This technique required a shorter decay period before counting than did beta counting and eliminated the detection of any ^{90}Sr contaminate. Standards were prepared by adding known quantities of ^{90}Y-labeled sand to nonradioactive fecal material.

Necropsies were performed on all sheep at death and on surviving sheep slaughtered 40 to 64 weeks postirradiation. Selected tissues were preserved in 10% buffered formalin for microscopic examination (detailed histopathology is reported elsewhere[15]).

RESULTS

Clinical Observations

Clinical signs of digestive disturbances were manifest in all sheep ingesting the synthetic fallout. Anorexia appeared between the fourth and tenth day after irradiation and continued in many of the sheep for several weeks (Fig. 1). There were no significant differences in severity of anorexia among the various GI-treatment groups; however, the duration of anorexia was less in sheep subjected to both GI and Skin irradiation. A significant interaction ($P < 0.05$) was observed among trials for feed intake, but this difference could not be correlated with the specific activity or the amount of sand fed. Feed intakes of all non-GI treatments did not differ from those of the control animals.

From minor to severe diarrhea was observed in the sheep after ingestion of ^{90}Y-contaminated feed. Fecal water began to increase 3 to 4 days after initiation of ^{90}Y feeding, reached a maximum at the fifth or sixth day, and then declined, probably as a result of the anorexia (Fig. 2). Another increase in fecal water, occurring between days 11 and 17, was not synchronous among all GI-treatment groups. The severe diarrhea was frequently accompanied by a slight mucous discharge and occasionally by a discharge of bright red blood, but hemorrhagic diarrhea was not evident. Marked changes in fecal water were not

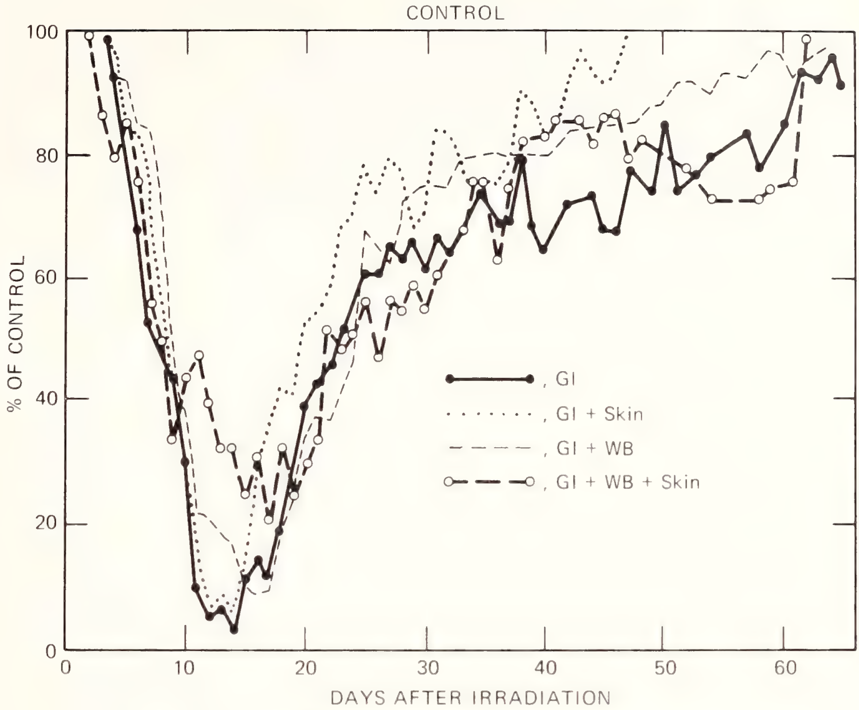

Fig. 1 Effect of gastrointestinal irradiation on feed consumption by sheep as a percent of feed consumption by control sheep. Feed intake of sheep receiving whole-body (WB) gamma and skin irradiation did not differ from that of control sheep.

observed in sheep of the non-GI-treatment groups during the 3-week period, nor was diarrhea a frequent occurrence among the surviving sheep after 3 to 4 weeks.

A marked increase in both water consumption and urine excretion ($P < 0.05$) was also associated with the severe illness of the GI-treatment groups (Table 1). The WB-treatment group also showed a less pronounced drop in water intake and urine excreta. However, no significant change in percentage of body water per kilogram of body weight as measured by tritium dilution was observed in a study using many of these animals (unpublished data).

An increase in body temperature was frequently observed in sheep of the GI-treatment groups, but this condition was neither continuous nor consistent. Pyrexia, however, usually was observed prior to death.

The changes in body weight during the 10-week period after irradiation are shown in Fig. 3. By the second week all the GI-treatment groups had lost approximately 20% of their initial body weight; this was probably a reflection of the severe anorexia and diarrhea. The animals receiving the triple insult continued losing weight; in this they differed significantly ($P < 0.05$) from the

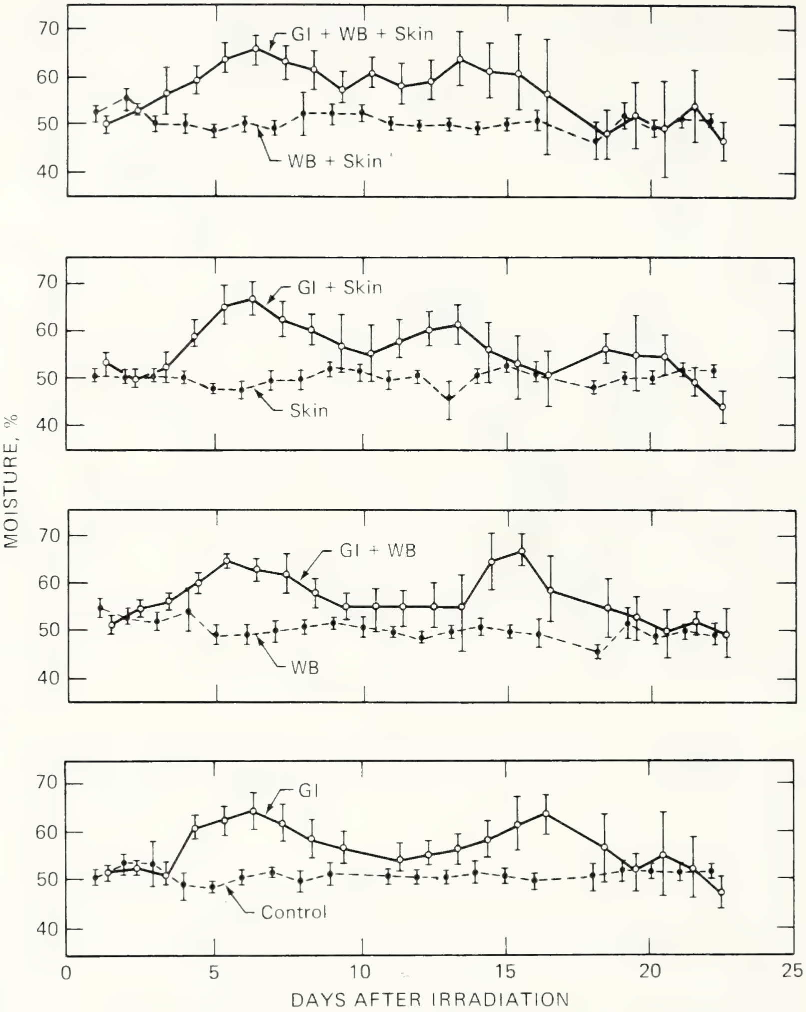

Fig. 2 Effect of irradiation on the moisture content of fecal excreta. Diarrhea occurred only in GI-irradiated sheep.

other GI-treatment groups by the fifth week. A sharp increase in body weight of the GI, GI + Skin, and GI + WB groups occurring between days 24 and 35, synchronous with the partial recovery in appetite, was probably a reflection of rumen fill.

Skin and Skin + WB irradiated sheep were unable to maintain their body weight on the restricted ration and lost over 10% of their weight by the seventh week. The WB-irradiated and control animals nearly maintained initial weight during this period of feed restriction. During the recovery period of ad libitum

Table 1

EFFECTS OF VARIOUS RADIATION TREATMENTS ON DAILY WATER CONSUMPTION AND URINE EXCRETION BY SHEEP

Treatment	Days after treatment				Days after treatment			
	1 to 6	7 to 12	13 to 18	19 to 24	1 to 6	7 to 12	13 to 18	19 to 24
	Water, ml/day*				Urine, ml/day*			
Control	929 ± 163	1206 ± 139	1180 ± 173	1257 ± 153	689 ± 78	723 ± 38	776 ± 44	804 ± 54
WB	752 ± 142	943 ± 167	754 ± 136	870 ± 137	617 ± 60	441 ± 38	341 ± 36	385 ± 42
Skin	680 ± 131	1003 ± 156	932 ± 147	1021 ± 52	592 ± 37	566 ± 40	625 ± 34	695 ± 38
GI	871 ± 149	283 ± 82	252 ± 625	596 ± 108	515 ± 40	371 ± 34	346 ± 23	361 ± 41
WB + Skin	877 ± 125	1315 ± 139	997 ± 117	1286 ± 134	563 ± 23	641 ± 17	667 ± 20	636 ± 21
GI + Skin	838 ± 154	732 ± 72	619 ± 180	869 ± 139	608 ± 30	379 ± 23	371 ± 26	417 ± 29
GI + WB	885 ± 148	930 ± 94	308 ± 86	866 ± 66	676 ± 59	863 ± 64	776 ± 56	876 ± 68
GI + WB + Skin	672 ± 128	550 ± 105	261 ± 67	520 ± 84	496 ± 52	341 ± 19	418 ± 28	422 ± 27

*Mean values ± standard error.

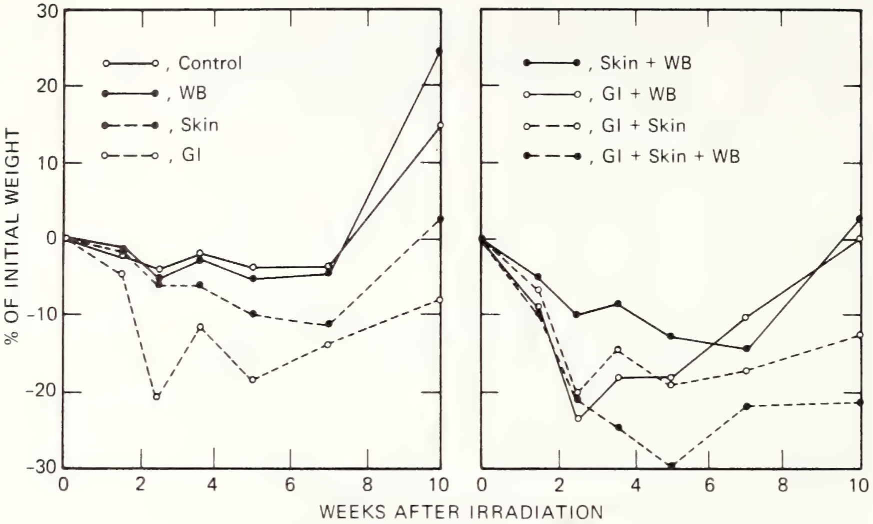

Fig. 3 Effects of irradiation on body weight (expressed as a percentage of the initial weight) of sheep fed a restricted diet for 7 weeks and then fed ad libitum.

feeding, all surviving animals gained weight. Survival weight at 40 weeks (Table 2) was significantly ($P < 0.05$) lower than that of control sheep for all treatments except WB, and the weight gain of GI + Skin and GI + Skin + WB groups was significantly less ($P < 0.05$) than that of all other treatment groups.

Table 2

EFFECT OF GI, WB, AND SKIN IRRADIATION ON SURVIVAL OF SHEEP

Treatment	Initial weight, kg	Survival* weight, kg	Deaths	
			No.	Days postirradiation
Control	31.3	55.6^{a}†		
WB (240 R gamma)	31.1	56.5^{a}	1	61‡
Skin (57,000 rads beta)	31.1	47.5^{b}	3	55,‡ 114, 120
GI (2.4 mCi ^{90}Y/kg)	32.6	50.5^{b}	3	25, 102,§ 133§
WB + Skin	33.1	48.8^{b}	2	156, 239
GI + Skin	31.4	36.3^{c}	2	134,§ 172§
GI + WB	30.2	52.7^{b}	4	5, 17, 19, 68§
GI + WB + Skin	30.5	37.8^{c}	4	20, 30, 47, 61

*Forty weeks postirradiation.

†The values followed by the same letter (a, b, or c) are not different at the 5% level of significance.

‡Accidental death not attributable to radiation.

§Killed following the development of ruminal and/or abomasal fistulae.

^{90}Y Excretion and Dosimetry

Fecal ^{90}Y excretion levels (as a percentage of the total dose) increased rapidly and reached a peak by the third or fourth day (Fig. 4). After feeding of the fallout simulant was discontinued, fecal radioactivity declined with an effective half-time of less than 1 day. Ninety-nine percent of the ^{90}Y had decayed or had been excreted by 8 to 10 days after feeding. There were no significant differences in excretion among the various GI-treatment groups.

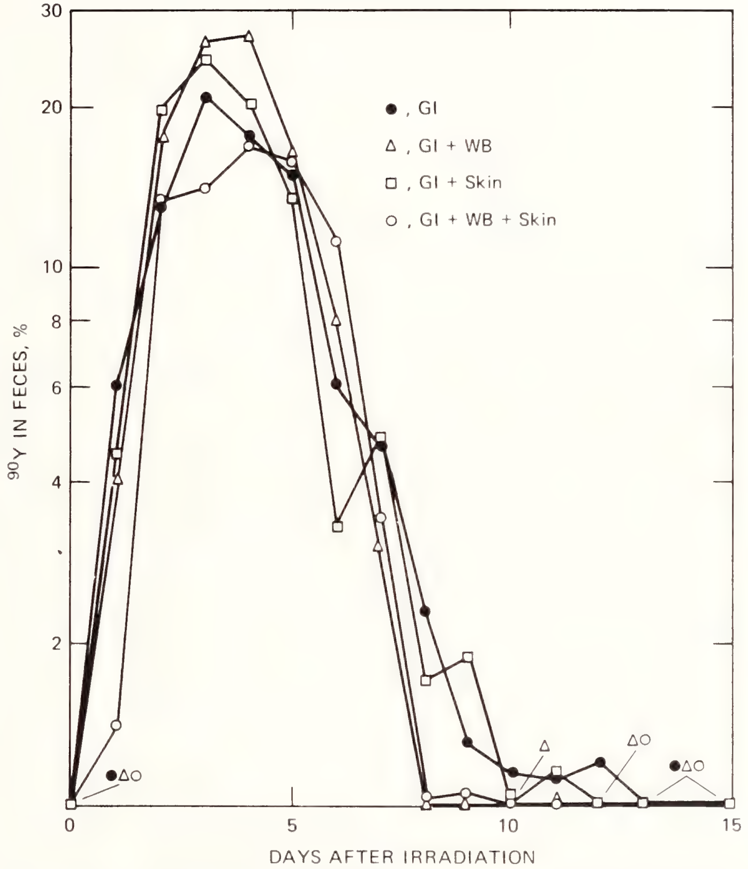

Fig. 4 Fecal excretion of ^{90}Y-labeled sand (percentage of total dose) fed for three consecutive days.

Radiophotoluminescent glass-rod dosimeters were used to estimate the absorbed dose from the ingested fallout simulant in 13 wethers of a similar weight and age.[14] The total dose, measured 7 to 10 days after initiation of feeding, was greatest in the fundic region of the abomasum (4.8 to 35 krads), a site of severe radiation damage. However, the doses measured in the affected areas of the rumen were only 0.5 to 5.3 krads and were not different from doses measured in the undamaged pyloric region of the abomasum (1.0 to 10.2 krads). This was probably due to the inability of the relatively large dosimeters to measure the dose delivered by the sand particles lodged among the papillae, rather than to a tissue-sensitivity effect.

Lethality and Gross Pathology

The number of deaths occurring in each treatment group and the number of days between irradiation and death are presented in Table 2. Early deaths occurred only in the GI-treatment groups, except for an accidental death of a Skin-irradiated sheep. Nearly 50% of the sheep receiving the two treatments involving a combination of GI and WB irradiation died within 60 days, a death rate significantly greater ($P < 0.01$) than the mortality from any of the other treatments.

Of the 24 sheep receiving Skin irradiation either as the only insult or in combination with GI or WB irradiation, six died between weeks 16 and 39. Four additional sheep were in poor condition at 40 weeks, but the remainder of the surviving Skin-irradiated sheep appeared to be healthy.

Abomasal prolapse through a hernial ring occurred in five sheep of the GI-treatment groups 68 to 172 days after treatment (Fig. 5a). In one sheep a small rumen fistula developed about 1 cm cranial to the prepuce 134 days after treatment, and a fistuous tract was seen in a sheep that died 60 days after irradiation. All these sheep were euthanatized due to their terminal condition.

The radiation damage to the gastrointestinal tract of the GI-treatment groups was similar to damage previously reported from ^{90}Y irradiation alone.[8] Major gastrointestinal lesions of sheep dying during the early period were usually confined to the ventral and lateral regions of the rumen and to the fundic–pyloric junction and associated laminae of the abomasum. The ventral and lateral regions of the rumen usually contained three to four areas of yellow polyplike fibrino-necrosis, which became friable and detached with time, leaving a smooth, pale, underlying base. By 40 to 60 days, tan or dark-colored scar tissue with a central erosion or necrosis was usually present. The abomasum was characteristically inflamed and edematous, with a large area of hemorrhagic necrosis at the caudal fundus and cephalic pylorus. The laminae were generally inflamed and edematous, and the pylorus was occasionally hyperemic and edematous. Only a slight increase in hemorrhage could be attributed to the added insult of WB irradiation. In several cases there were fibrino-hemorrhagic serosal adhesions of the abomasum and rumen to each other and/or to the abdominal wall. A purulent exudate was usually associated with the adhesions.

Damage to the intestines was limited to mild hyperemia and edema of the duodenal mucosa. Although the laminae of the omasum was congested in several sheep, necrosis of this organ was seen in only one sheep. Hydropericardium, dilatated cardiac ventricles, and heavy and edematous lungs were observed in these sheep at necropsy. Sheep of the GI-treatment groups surviving 40 to 52 weeks had residual ruminal and/or abomasal scars when slaughtered, and in many cases the scars contained eroded or necrotic centers as shown in Fig. 5b.

The locations of major damage in the gastrointestinal tract in these sheep differ from results predicted from dosimetric studies[6] in dogs and goats following an ingested dose of soluble ^{90}Y and studies[7] in sheep receiving lethal levels of soluble ^{144}Ce—^{144}Pr. The passage of sand particles through the rumen and abomasum appears to be independent of that of feed or fluids; thus sedimentation and concentration of these particles in the ventral portion of these organs resulted in significantly greater doses than expected from soluble material. In the intestinal tract the passage of sand in a homogeneous mixture with the less-fluid ingesta prevented settling of the particles and thus reduced the dose to the mucosa of the intestine.

Beta irradiaton of the skin produced erythema, cessation of wool growth, moist reaction of plasma exudate, and a gradual formation of a firm crusted mat of the wool during the first 4 to 6 weeks. The wool was easily removed if mechanically disturbed, but in most cases epilation was not complete until 10 to 16 weeks after irradiation (Fig. 6a). Along with epilation was sloughing of the epidermal layer leaving exposed a hemorrhagic necrotic dermal tissue. The healing and repair process was characterized by epithelialization of the periphery (2 to 4 cm) of the wound with the sequential development of an ivory horny or leaflike material. The central area of the injury of most sheep was still covered with necrotic tissue or a granulating surface when the sheep were slaughtered (Fig. 6b). The size of the irradiated area had decreased from 43 by 28 cm to approximately 25 by 16 cm. On one sheep retained for extended observation, 6- by 3-cm horny keratinizations about 3 cm thick developed by 62 weeks.

Hydropericardium, dilatated cardiac ventricles, and heavy edematous lungs were observed in Skin-irradiated sheep at death. However, milder manifestations of these abnormalities were common among Skin-irradiated sheep killed 40 to 64 weeks after irradiation.

The exact mode of death and the relation between the respiratory and cardiac involvement and the irradiation treatment of these sheep are not clear.

DISCUSSION

These results demonstrate that the additional stress of gastrointestinal irradiation injury from contaminated feed may cause not only a great loss of animal production but also a greater death rate than anticipated from WB irradiation alone. The early deaths were practically all due to WB and GI insults. The whole-body gamma LD_{50} of sheep at the dose rate used in the present study was approximately 200 rads (midline tissue dose).[1] However, when GI irradiation damage was imposed, the LD_{50} was reduced to 145 rads. With due regard for the limited sample size of this study, this is approximately a 25 to

Fig. 5a Mucosal surface of the abomasum of a sheep showing the fistula through which the lamina of the abomasum had prolapsed 14 weeks after the animal received ^{90}Y-labeled sand. Note the congested and hemorrhagic condition of the prolapsed tissue.

Fig. 5b Residual scar tissue in the rumen of a sheep 14 weeks after it received ^{90}Y-labeled sand. Similar scar tissue existed in all these animals slaughtered at 40 to 64 weeks. Note the necrotic center of the scar tissue.

Fig. 6a The irradiated area (43 by 28 cm) of a sheep's back 12 weeks after irradiation. Note the area of necrosis and the firm mat of undisturbed wool.

Fig. 6b The irradiated area (28 by 17 cm) of a sheep's back 40 weeks after irradiation. Note the ivory horny or leaflike material at the periphery, the nodular necrotic center, and the marked decrease in size of the irradiated area.

30% reduction in the LD_{50} from the whole-body gamma-ray dose. The mortality and secondary effects, such as loss of body weight, would certainly be critical to the livestock industry and would be of national importance as far as the food reserve is concerned in an emergency situation. A substantial radiation dose to the gastrointestinal tracts of livestock could result in reduction of meat production and reduced or lost milk production without causing death to the animal.

The additional stress of beta irradiation of the skin did not affect survival to a great extent for several months postirradiation. The loss in body weight was statistically significant ($P < 0.05$), but this might not have occurred if the ration had not been restricted. The large contiguous area of irradiated skin is probably an extreme situation, complete healing being virtually impossible. The fallout injury to the backs of the Alamogordo cattle was not uniform, and areas with minor or no injury probably influenced the healing of more severely affected areas.[3] The fact that major injury from skin irradiation was delayed may have allowed partial recovery from WB and GI trauma before the additional stress of skin irradiation was manifest. Thus skin injury from beta burns probably would not contribute significantly to sheep mortaility during the period immediately following a nuclear attack. However, this does not preclude possible effects of skin irradiation on longevity or other physiological mechanisms which can lead eventually to abnormal conditions.

Several deaths resulting from secondary effects occurred several months after irradiation. The development of hernias and fistulae would affect the sheep's longevity but not its value for food. However, accumulation in the meat of soluble fallout material such as ^{137}Cs and ^{90}Sr would be of concern. Most sheep with severely damaged skin could be used for food; few cases of liver abscesses or internal infection were apparent in these animals at death. During summer months vigilance was required in treating the injured skin to prevent severe damage from fly larvae. In winter the loss of heat from the damaged skin would be a problem and could affect the ability of these animals to grow or even to survive. The type of care necessary to prevent animal losses would be practically impossible to provide under range conditions. Nevertheless, in cases of food shortages, these survivors could still be sources of food if slaughtered prior to the onset of serious illness,[16] even though the meat quality and production per animal would probably be reduced.

Consideration must be given to the probability of animal exposure at the levels used in the present study. We can assume that a sheep must graze a pasture area of 6.8 m^2 to equal the daily feed intake of the sheep in this study and that 160 mCi/m^2 of gross fission products would be present at time H + 24 hr in any area having had an exposure rate of 100 R/hr at H + 1 hr.[17] Thus approximately 1100 mCi of fission products could be produced by time H + 24 hr on the area grazed by one sheep during a 24-hr period. The forage would have to retain only 7% of the fallout to produce the activity fed in the present study on day 1. Recent studies of retention of fallout sand indicate values at this level, but varying to some degree depending on the particle size, wind conditions, and pasture type and density.[18] Because of decay, the fallout arrival time would influence the amount of contamination at a given area, but, with due concern for all the variables involved, the activity fed in this study is considered to be a realistic level.

From the estimated exposures of the Alamogordo cattle,[3] a ratio of skin beta dose to whole-body gamma dose of 240 to 1 was used to determine the skin dose, but recent data indicate a beta-to-gamma ratio on plants of 12 to 1 from venting of underground nuclear devices.[19] A beta-to-gamma ratio of 10 to 1 was not sufficient to produce the severe effects observed in cattle exposed to beta irradiation.[3] This matter is probably not critical for postattack planning purposes, since even the high doses and the large areas of involvement in the present study did not affect animal survival for several months.

When predicting the vulnerability of farm livestock to fallout radiation, we must consider the effect of multiple radiation assaults on the survival and productivity of livestock. Many of our underground missile defense systems are located in areas of grazing livestock, and the possibility of surface nuclear attacks raises the question of the vulnerability of the livestock to fallout radiation.

ACKNOWLEDGMENTS

The UT–AEC Agricultural Research Laboratory is operated by the Tennessee Agricultural Experiment Station for the U. S. Atomic Energy Commission under Contract AT-40-1-GEN-242.

This work was supported by funds from the U. S. Office of Civil Defense and is published with the permission of the Dean of the University of Tennessee Agricultural Experiment Station, Knoxville.

REFERENCES

1. D. G. Brown, R. G. Cragle, and T. R. Noonan (Eds.), Proceedings of a Symposium on Dose Rate in Mammalian Radiation Biology, Apr. 29–May 1, 1968, Oak Ridge, Tenn., USAEC Report CONF-680410, UT–AEC Agricultural Research Laboratory, July 12, 1968.
2. D. G. Brown, Clinical Observations on Cattle Exposed to Lethal Doses of Ionizing Radiation, *J. Amer. Vet. Med. Ass.*, **140**: 1051–1055 (1962).
3. D. G. Brown, R. A. Reynolds, and D. F. Johnson, Late Effects in Cattle Exposed to Radioactive Fallout, *Amer. J. Vet. Res.*, **27**: 1509–1514 (1966).
4. C. F. Tessmer, Radioactive Fallout Effects on Skin. 1. Effects of Radioactive Fallout on Skin of Alamogordo Cattle, *Arch. Pathol.*, **72**: 175–190 (1961).
5. R. A. Conrad, The Effects of Fallout Radiation on the Skin, in The Shorter-Term Biological Hazards of a Fallout Field, Dec. 12–14, 1956, Washington, D. C., G. M. Dunning and J. A. Hilcken (Eds.), USAEC Report M-6637, pp. 135-141, U. S. Atomic Energy Commission and the Department of Defense, 1956.

6. M. M. Nold, R. L. Hayes, and C. L. Comar, Internal Radiation Dose Measurements in Live Experimental Animals—II, *Health Phys.*, **4**: 86–100 (1960).
7. M. C. Bell, Airborne Radionuclides and Animals, in *Agriculture and the Quality of Our Environment,* N. C. Brady (Ed.), pp. 77-90, Symposium No. 85, American Association for the Advancement of Science, Washington, D. C., 1967.
8. M. C. Bell, L. B. Sasser, J. L. West, and L. Wade, Jr., Effects of Feeding ^{90}Y-Labeled Fallout Simulant to Sheep, *Radiat. Res.*, **43**: 71–82 (1970).
9. H. Baxter, J. A. Drummond, L. G. Stephens-Newsham, and R. G. Randall, Reduction of Mortality in Swine from Combined Total-Body Radiation and Thermal Burns by Streptomycin, *Ann. Surg.*, **137**: 450–455 (1953).
10. L. A. George, Jr., P. L. Hackett, and L. K. Bustad, Triple Radiation Assault on Debilitated Lambs, USAEC Report HW-42540, General Electric Company, April 1955.
11. M. C. Bell and C. V. Cole, Vulnerability of Food Crop and Livestock Production to Fallout Radiation. Final Report, USAEC Report TID-24459, UT–AEC Agricultural Research Laboratory, Sept. 7, 1967.
12. J. S. Cheka, E. M. Robinson, L. Wade, Jr., and W. A. Gramly, The UT–AEC Agricultural Research Laboratory Variable Gamma Dose-Rate Facility, *Health Phys.*, **20**: 337–340 (1970).
13. M. C. Bell, Flexible Sealed ^{90}Sr–^{90}Y Sources for Large Area Skin Irradiation, *Int. J. Appl. Radiat. Isotop.*, **21**: 42–43 (1970).
14. L. Wade, Jr., R. F. Hall, L. B. Sasser, and M. C. Bell, Radiation Dose to the Gastrointestinal Tract of Sheep Fed an Insoluble Beta-Emitter, *Health Phys.*, **19**: 57–59 (1970).
15. J. L. West, M. C. Bell, and L. B. Sasser, Pathology of Gastrointestinal-Tract Beta-Radiation Injury, this volume.
16. J. H. Rust, Report of the National Academy of Sciences Subcommittee for Assessment of Damage to Livestock from Radioactive Fallout, *J. Amer. Vet. Med. Ass.*, **140**: 231–235 (1962).
17. National Academy of Sciences–National Research Council, Damage to Livestock from Radioactive Fallout in Event of Nuclear War, Publication 1078, Washington, D. C., Dec. 20, 1963.
18. J. E. Johnson and A. I. Lovaas, Deposition and Retention of Simulated Near-In Fallout by Food Crops and Livestock. Technical Progress Report No. 1, Report AD-695683, Colorado State University, May 1969.
19. W. A. Rhoads, R. B. Platt, R. A. Harvey, and E. M. Romney, Ecological and Environmental Effects from Local Fallout from Cabriolet. I. Radiation Doses and Short-Term Effects on the Vegetation from Close-In Fallout, USAEC Report PNE-956, EG&G, Inc., Aug. 23, 1968.

SIMULATED-FALLOUT-RADIATION EFFECTS ON LIVESTOCK

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ABSTRACT

Cattle ingesting ^{90}Y-labeled fallout simulant at the rate of 2 mCi/kg of body weight were more severely affected than those given 57,000 rads beta irradiation to 8% of the dorsal body surface. Whole-body irradiation of 240 R from ^{60}Co at 1 R/min affected only blood platelets and leukocytes. When these three treatments were combined on eight steers, all died within 54 days. Cattle were more sensitive to simulated-fallout radiation than sheep, but major damage from ingested radioactivity was in the rumen and abomasum of both species. No data were found on combined fallout-simulant effects on simple-stomach animals, but effects are predicted to be less than in ruminants. Sheltering cattle in barns would be the most effective practical measure to increase animal survival and reduce productivity losses in the survivors. Corralling animals to prevent their grazing heavily contaminated pastures would be an alternative where barns are not available. About 80% of the 112 million U. S. cattle are on pasture. In a 4-hr roundup time, it is estimated that this percentage could be reduced to 34% by corralling about 43 million cattle and by placing about 31 million in barns.

In the event of nuclear war, major farm livestock losses from airbursts would be caused principally by blast and thermal injury, whereas losses from surface bursts would be caused by fallout-radiation injury. Airbursts would be expected to be concentrated on urban areas and would not involve a large number of livestock, but fallout from surface bursts would probably include areas with heavy livestock populations. Grazing livestock would be exposed to gamma radiation to the entire animal, beta radiation to the skin, and beta radiation to the gastrointestinal tract. Most of the gamma exposure would come from ground fallout, but the total exposure would include the gamma component of fallout ingested and also from particles retained on the skin.

Early reports[1] indicated that beta irradiation was of little consequence in affecting livestock survival and production, but more-recent data show that, owing to stratification of simulated fallout particles in the gastrointestinal tract, beta irradiation can severely affect survival and productivity of sheep.[2,3] The early reports, based on dosimeter readings in dogs and goats fed soluble ^{90}Y, have recently been reconfirmed by Ekman, Funkqvist, and Greitz,[4] who fed goats soluble ^{153}Sm and ^{140}La.

The purpose of this paper is to 'report the effects of simulated-fallout radiation on yearling beef calves and to predict the impact of fallout radiation on the livestock industry.

EXPERIMENTAL PROCEDURE

Sixty-four yearling Hereford steers averaging 184 kg were divided into eight groups and randomly assigned to the treatments listed in Table 1. Bilateral exposure to whole-body gamma (WB) irradiation of an air dose of 240 R was made at a dose rate of 1 R/min with a ^{60}Co facility.[5] Whole-body exposure was made 12 to 20 hr before the initiation of the other treatments. Exposure of about 8% of the body surface[6] (Skin) to beta irradiation was accomplished by placing two flexible sealed ^{90}Sr—^{90}Y sources[7] over the thoracolumbar region to give 57,000 rads at the surface of the hair at the rate of 17 to 25 rads/min. Gastrointestinal (GI) irradiation was accomplished by feeding 2 mCi of ^{90}Y-labeled sand per kilogram of body weight using the previously described procedure.[2] In addition to these three treatments and all possible combinations

Table 1

RADIATION-TREATMENT EFFECTS ON WEIGHTS AND SURVIVAL OF YEARLING CATTLE

Treatment	Weights, kg		Deaths	
	Initial	After 5 weeks	No.	Days after treatment
Control	183.4* ± 6.9	198.9 ± 6.6	0	
WB	183.5 ± 5.9	193.5 ± 6.1	0	
Skin	186.4 ± 6.5	193.9 ± 6.2	0	
GI	184.3 ± 4.9	149.4 ± 6.5	3	14, 44, 61
WB + Skin	183.6 ± 5.7	189.1 ± 4.2	1	168
GI + Skin	184.8 ± 2.2	145.1 ± 4.4	4	25, 53, 67, 83
GI + WB	185.5 ± 4.9	141.5 ± 3.5	5	14, 17, 19, 40, 54
GI + WB + Skin	183.5 ± 5.3	135.5 ± 9.5	8	15, 19, 19, 25, 25, 27, 33, 54
Starved control	171.7 ± 7.4	155.7 ± 5.3	0	

*Mean values ± standard error.

of treatments, there was a control group and a group whose feed was restricted to that consumed by the GI group. One animal was exposed to each of the treatments at a time with eight replications over a period of 11 months. During a period of adjustment before treatment and for 5 weeks thereafter, the cattle were kept in individual stalls[8] for separation and collection of urine and feces. During this time they were daily fed 2.7 kg of alfalfa pellets moistened with 0.8 kg of water. The ^{90}Y-labeled sand was mixed with the moistened alfalfa for each animal for three consecutive days. The ^{90}Y averaged 9.4 mCi/g of sand (88 to 175 μ) at the time of feeding. Steers weighing 184 kg were fed 368 mCi of ^{90}Y in 39 g of sand on day 1; this quantity had decayed to 284 mCi by day 2 and to 219 mCi by day 3. Control animals were fed the same quantity of nonradioactive sand for each of the 3 days. Feed intake, body temperature, and signs of radiation injury were recorded daily.

After 5 weeks of close observation in the collection stalls, the steers were grouped together by trial in large pens with shelter, access to limited pasture, and free access to grass hay, water, and trace-mineralized salt. In addition, they were fed enough 15%-protein grain mixture to provide a growth rate of about 0.4 kg daily for the control animals. Body weights and general recovery were observed periodically for 40 weeks after treatment.

In addition to these treatment groups, four yearling Hereford steers of comparable size and origin were implanted with glass-rod dosimeters into several segments of the gastrointestinal tract by a previously described procedure.[9] After 3 weeks they were fed 2 mCi ^{90}Y sand for 3 days. They were subjected to necropsy 13 days later, and the recovered dosimeters were read.

Necropsy examinations were performed on all dead animals, and specimens of selected tissues were photographed and then preserved in 10% formalin for histological examination.

RESULTS

Table 1 shows that deaths occurred only in treatment groups including GI irradiation, with the exception of one steer that died 168 days after WB and Skin irradiation. Of the 20 deaths, 17 occurred within 60 days after treatment, and only 7 of the 17 occurred within 30 days. From these data it appears more reasonable to use $LD_{50/60}$ than $LD_{50/30}$ for grazing cattle exposed to combinations of fallout exposures.

Most of the early deaths were associated with combinations of GI and WB exposures with the resulting hemorrhagic necrotic involvement. Damage in the four major "pockets" of the rumen was more extensive than was observed in sheep. The rumen floor contained large fibrinous masses. In addition, sections of the ventral reticular honeycombs of most of the cattle were filled with a rubbery, yellow, glandular-appearing material. Minor fibrinous necrotic areas were seen in the omasum of most of the steers. Major areas of hemorrhagic necrosis were surrounded by edematous hyperemic laminae in the abomasum of all cattle fed ^{90}Y. Adhesions among the rumen, abomasum, and reticulum were frequent, and some involved a mass of gelatinous serosal exudate. Gross lesions in the large intestine were restricted to minor areas in the cecum and colon of a few steers fed ^{90}Y sand. Several animals showed degenerative changes in the heart. Necropsy results are given in more detail in an accompanying paper.[10]

Data summarized in Table 1 also show that the combinations of radiation sources were more detrimental than single exposures not only to survival but also to body weight of the animals at 5 weeks after exposure. No animals given the combined GI + Skin + WB irradiation treatments survived longer than 54 days. At 35 days the three surviving steers had lost an average of 48 kg, which was the greatest loss by any treatment group. Only the "starved" control steers and the steers fed ^{90}Y sand lost weight. Although feed intake by the starved controls was restricted to that of the GI-treated steers, the GI-treated steers lost 25% of body weight, while the starved controls lost 9% and the normal controls gained 8%. The excess weight loss by the GI-treated steers was probably due to pyrexia and mild-to-severe diarrhea.

The depression in feed intake by the GI-treated steers was dramatic, but only minor differences were noted among the four groups fed ^{90}Y sand (these data are pooled in Fig. 1). After 9 days, feed intake averaged less than 5% of the controls for the remainder of the 28-day period of observation. Comparable data on sheep, also shown in Fig. 1, indicate that depression of feed intake occurred later and that appreciable recovery was evident by day 28. Feed consumption by cattle and sheep receiving WB, Skin, and WB + Skin treatments was not different from the untreated control animals for each respective species.

Since all cattle were group fed after 28 days of individual feeding, no feed data are available on the treatment groups after that time. Observations on the surviving cattle are incomplete at this writing, but the 40 weeks of observations

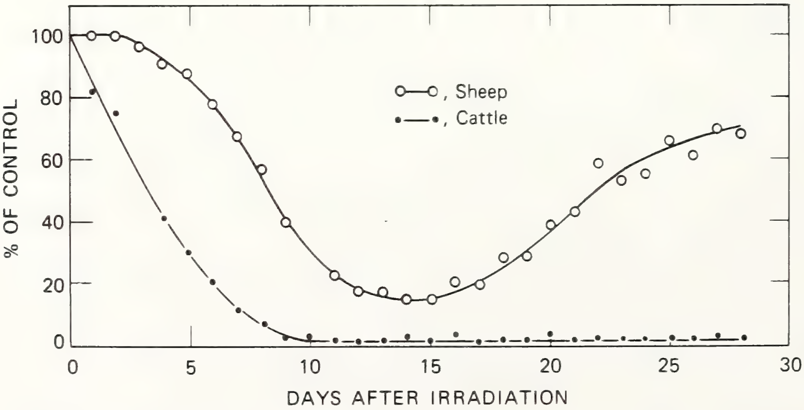

Fig. 1 Feed consumption by sheep and cattle fed ^{90}Y-labeled fallout simulant. Feed consumed by WB- and Skin-irradiated animals was the same as that consumed by controls.

are complete on four of the eight replications. During this period the average kilograms of weight gained per surviving animal for each treatment group were: control, 118; WB, 131; Skin, 66; GI, 48; Skin + WB, 58; GI + WB, 36; and GI + Skin, 22. None of the animals receiving GI + WB + Skin treatment survived beyond 54 days (Table 1). These data show that GI-treated survivors had regained much of the weight lost in the first 28 days (Table 1).

Body temperature was not significantly different among the controls, WB, Skin, and WB + Skin treatment groups for the 25-day postexposure period. All cattle fed ^{90}Y-labeled sand showed elevated body temperature, which persisted longer in those with combined GI and WB irradiation. The starved control group showed a drop in body temperature, indicating a lowered metabolic rate (Table 2).

Except for the larger exposure area, the skin irradiation changes developed similarly to those described by George and Bustad.[11] A moist reaction developed during the first 3 weeks, with crusted plasma and epilation in 8 to 12 weeks, followed by a hemorrhagic necrosis.

Whole-body gamma irradiation of 240 R at 1 R/min alone did not give the characteristic visible signs of radiation sickness. These animals did show the depression of white blood cells and platelets.

All steers fed ^{90}Y-labeled sand had mild-to-severe watery diarrhea. The onset of diarrhea varied from 6 to 15 days after initiation of the ^{90}Y feeding. In about half of the animals, this was followed by regurgitation of feed and water. Also about half of the animals were audibly grinding their teeth constantly. The loss of body fluids from diarrhea and vomiting probably contributed to the death of many of these animals.

DISCUSSION

General

The results of these investigations on simulated-fallout-radiation effects on beef cattle are similar to the data obtained on sheep.[3] Nevertheless, there were differences in response between the two species which would prevent the exclusive use of sheep as models for beef cattle. Both species are grazing ruminants with many similar physiological functions, but they differ in size and grazing habits.

These data clearly demonstrate that cattle exposed to simulated-fallout grazing conditions were so severely affected by the combination of treatments that there were no survivors at nonlethal levels of WB exposure where no physical signs of radiation sickness were seen from WB exposure alone.

Skin Exposures

No deaths occurred from Skin exposure alone, but, in combination with other treatments, Skin exposure apparently contributed to increased mortality

Table 2

BODY TEMPERATURE OF CATTLE EXPOSED TO RADIATION TREATMENTS

Treatment	Days after irradiation				
	1 to 5	6 to 10	11 to 15	16 to 20	21 to 25
Control	101.9* ± 0.2	101.8 ± 0.2	101.7 ± 0.1	101.6 ± 0.2	101.9 ± 0.2
WB	102.1 ± 0.2	102.0 ± 0.2	101.7 ± 0.2	102.1 ± 0.2	102.1 ± 0.2
Skin	101.4 ± 0.2	101.7 ± 0.1	101.7 ± 0.1	102.0 ± 0.2	101.6 ± 0.2
WB + Skin	101.5 ± 0.2	101.7 ± 0.1	102.0 ± 0.1	102.4 ± 0.2	102.1 ± 0.2
GI	101.9 ± 0.3	103.0 ± 0.3	102.9 ± 0.2	102.1 ± 0.2	101.6 ± 0.2
GI + Skin	102.0 ± 0.2	103.2 ± 0.2	103.3 ± 0.2	102.4 ± 0.2	101.6 ± 0.2
GI + WB	101.4 ± 0.2	102.4 ± 0.2	104.4 ± 0.3	104.1 ± 0.4	103.2 ± 0.2
GI + WB + Skin	102.1 ± 0.2	102.4 ± 0.2	104.0 ± 0.3	104.6 ± 0.2	103.9 ± 0.2
Starved control	101.6 ± 0.2	101.1 ± 0.1	100.9 ± 0.2	101.2 ± 0.3	100.5 ± 0.2

*Mean Fahrenheit temperature ± standard error.

rates. Although the flexible, sealed sources exposed rectangular areas of 28 by 43 cm fairly uniformly, these areas resembled the beta-damaged areas on the Alamogordo cows.[12] Healing around the edges reduced the severely damaged area by the end of 40 weeks of observation. No data are available on the dimensions of the original damaged areas of the cattle exposed in 1945, but in 1950 hyperkeratosis was evident from the anterior withers to the tail head and extended up to about 23 cm laterally from the midline of one of these cattle. Some areas of extensive hyperkeratotic plaques and horns measured on the preserved hide taken from the same cow in 1960 were 13 by 10 cm with an elevation of about 2 cm over most of this surface. The skin exposure of the Alamogordo cattle was not uniform, but apparently some of these areas could have originally been as large and the damage as extensive as those seen on our cattle from the exposed rectangular areas of 28 by 43 cm. Some healing and tissue repair is already evident in the Skin-irradiated areas on the cattle, but the extensive hyperkeratosis has not developed in those exposed in July 1969. A few areas of moderate hyperkeratosis and scaling have developed.

Frequent insecticide spraying was required to reduce the fly problem on the skin-damaged areas during warm weather. Since these cattle had free access to shelter and shade, exposure to weather extremes was considerably reduced. Animals in other areas of the United States could be exposed to greater climatic extremes, and many would have much less protection. The loss of the dorsal hair coat covering 8% of the body surface would be expected not only to increase thermal losses but also to increase nutrient requirements for tissue repair. This is evident by the limited data showing that the Control steers gained 52 kg more than the Skin-irradiated steers during the 40 weeks of observation.

GI Exposure

Feeding steers 2 mCi of ^{90}Y sand per kilogram of body weight was more detrimental than feeding 2.4 mCi/kg to sheep. This was reflected in greater reduction of feed consumption, increased mortality, and increased organ damage. The reduction in feed intake was accompanied by a more severe diarrhea, vomiting, and grinding of teeth. Fallout-simulant feeding was calculated to represent a 9% forage retention with the calculation procedure described previously.[1] Since this corresponds closely to the level of 7% calculated for sheep,[2] the results were expected to be quite similar. Possibly cattle are more sensitive to GI beta irradiation, or perhaps the larger accumulation of ^{90}Y-labeled sand in the damaged areas produced a greater exposure. Dosimetry data are incomplete, but preliminary data indicate that the rumen exposure was greater than that observed in sheep.[9]

The long-term effects of GI exposure in cattle survivors appear to be less than in sheep. None of the surviving cattle developed rumen fistulae or abomasal hernia and prolapse, but six sheep fed ^{90}Y sand developed these sequelae. The greater thickness of cattle tissue probably reduced the eventual extent of injurious effects on tissues adjacent to the primary site of injury, and no adhesions were found between affected organs and the abdominal wall.

These data show that feeding a particulate fallout simulant of size and density similar to early fallout produces results quite different from using soluble fallout simulants.[4,13] Early fallout particles would be expected to collect in pockets in the gastrointestinal tract of ruminants as shown in this and similar studies.[2,3]

WB Exposure

No cattle died from exposure to 240 R at 1 R/min unless this treatment was used in combination with other radiation exposures. Except for depressed white blood cells and blood platelets, none of these steers showed the depressed appetite and other symptoms of radiation sickness described by Brown.[14] Brown established an $Ld_{50/30}$ of 543 R in a study of 70 adult female Hereford cattle exposed to 450 to 700 R at 0.9 R/min; about 10% of the cattle exposed to 450 R were lost. More-recent unpublished data from the same laboratory show a loss of five of 120 Hereford heifers exposed to 300 R at 0.7 R/min and no losses from 200 R exposure. None of these deaths occurred during the second 30 days after exposure, but four of the eight deaths from a combination of WB + GI + Skin exposures were observed in the first 30 days and the other four during the second 30-day period.

Animals surviving the WB component of fallout exposure of 240 R alone would be expected to produce almost as well as nonirradiated animals. During the 40 weeks of observation, the weight gain of the four WB-irradiated cattle averaged 131 kg, while the controls gained 118 kg. Data on other animals indicate life-shortening WB-irradiation effects, but, when aged cattle cease producing or production becomes uneconomical, they are normally culled and replaced by young breeding stock.

Combined Effects

Although no cattle died at a WB exposure of 240 R and all died from a combination of WB + GI + Skin, there are no data available for cattle on what might be expected from a different forage-retention level or from other combinations of exposures. It would be prohibitively expensive to obtain data on all possible combinations, but the need for more data is clearly indicated, and threshold lethality levels should be determined. These data show that combinations of two or more radiation injuries are lethal to a greater percentage of animals and severely affect productivity of survivors. Whole-body exposures affect the bone marrow as the most sensitive target system, and beta exposure to the skin and gastrointestinal tract affects the local tissue primarily, but abscopal effects are also observed on mineral metabolism.[2] Whole-body gamma radiation from ^{60}Co is reduced by 50% in about 18 cm of unit-density tissue, whereas beta penetration from ^{90}Y is reduced by 50% by a thickness of only 1 mm of unit-density tissue.

IMPLICATIONS

Livestock Inventories

Since the 1967 report on livestock and postattack recovery,[15] the inventory and productivity of the major classes of livestock have increased. Cattle number above 112 million and supply over 50 kg of meat and over 150 kg of dairy products per person in the United States annually. Production and consumption of pork and poultry products have also increased, With the increase in the livestock inventories, the estimated market value for cattle alone has now increased to over $20 billion. This is indeed a food reserve worth evaluating in terms of reliable vulnerability estimates for fallout effects on survival and production of these animals. Cattle can produce highly nutritious food when fed products not usable for human consumption. However, if 90% of the breeding cattle were lost, about 11 years would be required to replenish the inventory of breeding animals;[15] this further emphasizes the need to consider vulnerability and protective measures. In contrast, the inventory of poultry and swine is small; about 1 year is required to replenish a 90% loss of breeding stock.[15] In even greater contrast is the radiation resistance and small inventory of seed grains required to resume normal production of food crops. These food crops are sensitive to fallout radiation only during the growing season, but livestock are sensitive at all seasons of the year.

The importance of livestock production in helping to improve world protein supplies has been reemphasized by Director General Boerma of the Food and Agriculture Organization of the United Nations in a new "Indicative World Plan." In the short run, he recommended that swine and poultry production be increased and that in the more distant future ruminant livestock inventories be built up to provide more meat and milk. Recommendations were made also to simultaneously increase production of cereals and crop products in the developing nations.[16]

Loss Predictions

In estimating survival of livestock populations in a nuclear war, most builders of damage-assessment models have used gamma radiation as the only criterion. Some estimate that, under the same conditions, half the human deaths will result from causes other than gamma irradiation. Soft targets, such as major cities, would probably get mostly airbursts, which would cause many thermal and blast fatalities among the population. Hard targets would be expected to be hit by surface bursts, which increase the fallout fatalities. Livestock are widely dispersed and would be affected mostly by fallout from surface bursts. Nevertheless, some losses would occur around population centers. In 1969 the livestock yards in Chicago, Ill., handled 1.1 million cattle and 1 million hogs; those in Omaha, Nebr., handled 1.5 million cattle and 1.8 million hogs.[17] Although marketing is being decentralized, many livestock are in transit through large population centers in addition to those destined for slaughter.

The limited data available in this and the preceding paper[3] show definitely that, regardless of the conclusions based on dosimeter readings in animals fed soluble radioisotopes, grazing livestock losses from fallout radiation would not be limited to gamma irradiation alone. The 1970 Swedish paper[4] based on dosimeter readings in goats given a solution of ^{153}Sm and ^{140}La neglects the physical characteristic of fallout particles in combination with the physiological functions of the ruminant gastrointestinal tract. Fallout particles from a surface nuclear burst deposited downwind on forage in an area where the gamma exposure would be above 200 R would be expected to collect in "pockets" in the rumen and abomasum owing to the strong muscular movements of the different compartments of these organs. This has been demonstrated not only by recovery of sand particles but also by observation of damaged areas and by dosimetry measurements. Radiation irritation to the colon would be expected to reduce the further beta exposure by increasing the rate of passage and by reducing water reabsorption in the lower large intestine. The reports from dosimeter readings in dogs and goats[4,13] are from levels of soluble isotopes which showed minor to no physiological responses. Soluble isotopes would be expected to adsorb to feed particles and move in a homogeneous mixture with the ingesta. Early fallout levels apt to affect livestock survival would not be expected to have a solubility above 10%, but radiations from ^{153}Sm and ^{140}La appear to be characteristic of beta and gamma-ray emissions of mixed fission products. For animal research the gamma radiation from ^{153}Sm and ^{140}La would increase the hazard to personnel using these isotopes to label fallout-simulant sand particles, but the beta energy would be more characteristic of early fallout than that from ^{90}Y used in most other studies.

Data available on grazing livestock indicate that cattle are the most sensitive species to combinations of fallout exposures. Therefore damage-assessment estimates should concentrate on cattle since they supply more food products and require more time to replenish breeding stock than any other U. S. food source. Since there were no losses of cattle exposed to 240 R of gamma radiation but there was 100% loss of those exposed to 240 R of WB + GI + Skin irradiation, it is difficult to estimate the $LD_{50/60}$ gamma exposure when combined with the beta exposure. Based on the limited data available, very rough estimates of $LD_{50/60}$ exposures for livestock in barns and corrals or pens and for those grazing heavily contaminated pastures are presented in Table 3. Data on sheep represent a 7% forage retention of fallout with the combined effects being lethal to four of eight animals;[3] data on cattle are for 9% forage retention with a loss of eight out of eight exposed animals. Apparently differences between these species are greater than can be accounted for by forage-retention differences. No data are available on cattle consuming forage at

Table 3

ESTIMATED LIVESTOCK LETHALITY ($LD_{50/60}$) FROM FALLOUT-GAMMA-RADIATION EXPOSURE ALONE AND IN COMBINATION WITH BETA RADIATION

	$LD_{50/60}$, total gamma exposure, R		
	Barn (WB)	**Pen or corral (WB + Skin)**	**Pasture* (WB + Skin + GI)**
Cattle	500	450	180
Sheep	400	350	240
Swine	640	600†	550†
Equine	670	600†	350†
Poultry	900	850†	800†

*Assumed forage retention of 7 to 9%.

†No data available.

the extremes of 5 to 25% forage retention reported by the Colorado workers[18] using 88- to 175-μ sand. Also, no data are available on the effects of smaller radioactive fallout-simulant particles on the gastrointestinal tract of sheep or cattle.

Estimates in Table 3 on combined effects on swine, equine, and poultry were obtained, not from research results, but from estimates based on grazing habits and on gastrointestinal anatomical and physiological functions of these species.

To determine the number of animals which might be exposed, we can make assumptions on the different management practices for the classes of livestock within each species. A very rough estimate has been made of the normal numbers of the 112 million cattle expected to be on pasture, in penned or corralled areas, and in shelters (Table 4). The 4-hr roundup time does not imply that livestock producers would neglect other emergency procedures to protect livestock, but only what might be done in 4 hr to help protect cattle.

Removal from pasture offers the greatest protection to grazing livestock, as shown in Table 3. Pastured dairy cows are normally near the milking parlors and would be much easier to confine than other cattle. Milk cows and some calves creep-fed on pasture would get supplemental grain, and thus their intake of radioactive fallout would be diluted, but almost all other grazing cattle would depend entirely on pasture forages and mineral supplements. It would be futile to attempt to corral animals in the large range cattle operations in a short time, and 4 hr is insufficient time for many range operations. The operators of small family farms, which are typical of most of Tennessee farms, would be able to confine cattle in a short time. For this reason the surveys by Griffin[19] are more optimistic than the data presented in Table 4. His pilot survey covered 176 farms in Tennessee, but no data were found for the entire United States. Again it should be emphasized that the greatest reduction in the number of lethalities can

Table 4

ESTIMATED NUMBERS OF U. S. CATTLE SHELTERED OR CORRALLED INITIALLY AND AFTER A 4-HR ROUNDUP EFFORT

	Number in millions			
	Milk cows	Feedlot cattle	Other cattle	Total
Shelter				
No warning	3	< 1	1	5
4-hr roundup	8	3	20	31
Pen or corral				
No warning	4	10	4	18
4-hr roundup	5	8	30	43
Pasture				
No warning	7	1	81	89
4-hr roundup	1	< 1	36	38
Total	14	12	86	112

be made by preventing livestock from grazing for the first few days after fallout arrival (Table 3).

Productivity of Survivors

WB Effects

Gamma irradiation alone had no effect on rate of weight gain of cattle surviving exposure to 240 R at 1 R/min given as discussed previously, and no measurable effect was seen on the sheep exposed to the same treatments, as reported by Sasser, Bell, and West.[3] Differences were not statistically significant, but WB-irradiated sheep gained 25 kg in 40 weeks, whereas controls gained 24 kg. These data are in agreement with earlier reports on swine,[20] minor effects on milk production,[21,22] and minor effects on poultry.[23]

Reproductive performance has not been affected in 179 surviving beef cows covering 8 years after an acute WB exposure, and offspring performance has not been different from control performance.[24] Embryos of food-producing animals exposed to a minimum of 100 R are sensitive to bone deformities for only 3 days during the first trimester of pregnancy.[24,25]

WB + Skin Effects

Livestock surviving in open pens, corrals, and feedlots could receive sufficient exposure to affect productivity. The skin exposures of the cattle discussed previously and those of sheep[3] were sufficient to reduce weight gains, and the exposure levels were quite similar to those reported for the nonlethal exposure of cattle at Alamogordo in 1945.[12] Skin-irradiated sheep gained 16 kg in 40 weeks; controls gained 24 kg. Observations are incomplete on cattle, but the 40-week gains (in kilograms) on four animals per treatment were: control, 118; WB, 131; Skin, 66; and Skin + WB, 58. All these animals had access to shelter; greater thermal losses would be expected under more-extreme environmental conditions. Skin irradiation at levels causing alopecia would also be expected to reduce milk production and increase problems from external parasites, which could also lower productivity and reduce survival.

WB + Skin + GI Effects

Livestock ingesting sufficient radioactive fallout to elicit a physiological response would almost always be expected to be exposed to WB and Skin irradiation levels sufficient to cause physiological changes. The four sheep surviving a combination of these three treatments recovered from the early weight loss, but the net 40-week gain was only 7 kg compared with 24 kg for the eight controls. No cattle survived a combination of these three treatments. Observations are continuing on the survivors of GI exposure alone and in combination with either Skin or WB. The conclusion is therefore made that grazing ruminants surviving in a fallout field where the gamma exposure is above 100 R would suffer a large reduction in productivity. No data are available on simulated exposure of grazing simple-stomach livestock, but effects would probably be less than in grazing cattle and sheep.

Protective Measures

Ideally, fallout shelters with high protection factors would save the most livestock from radioactive fallout in the event of a nuclear war. From a practical viewpoint, existing barns providing a protection factor of 2, as shown in the limited survey by Griffin,[19] would offer protection much greater than that from the reduction in gamma exposure alone. Cattle in barns would probably survive a gamma contour (measured 1 m above the ground in the open) ten times greater than cattle grazing on pasture.

Cattle restricted to a small area with a high density of animals and limited or no grazing opportunity would have a much better chance of survival than those on pasture. They would provide mutual shielding against gamma exposure and, more important, would not receive the high-level GI exposure. In the USSR[26] it was suggested that canvas and blankets may be used to protect the skin of large animals. Also suggested was a chemically treated protective muzzle bag to be used on cattle to reduce inhaled fallout and to prevent them from eating contaminated feed.

Prevention of grazing of contaminated pastures for the first few days is one of the major ways of reducing lethality and productivity losses. Under these conditions, giving cattle and sheep no feed at all is much better than permitting them to graze heavily contaminated pastures. Farm livestock can survive many days without feed but only a few days without water. Providing water for animals in barns and/or pens would be an additional problem.

ACKNOWLEDGMENTS

The UT–AEC Agricultural Research Laboratory is operated by the Tennessee Agricultural Experiment Station for the U. S. Atomic Energy Commission under Contract AT-40-1-GEN-242.

This work was supported by funds from the U. S. Office of Civil Defense and is published with the permission of the Dean of the University of Tennessee Agricultural Experiment Station, Knoxville.

REFERENCES

1. National Academy of Sciences–National Research Council, Damage to Livestock from Radioactive Fallout in Event of Nuclear War, Publication 1078, Washington, D. C., Dec. 20, 1963.
2. M. C. Bell, L. B. Sasser, J. L. West, and L. Wade, Jr., Effects of Feeding ^{90}Y-Labeled Fallout Simulant to Sheep, *Radiat. Res.*, **43**: 71–82 (1970).
3. L. B. Sasser, M. C. Bell, and J. L. West, Simulated-Fallout Radiation Effects on Sheep, this volume.
4. L. Ekman, B. Funkqvist, and U. Greitz, Beta- and Gamma-Dose Measurements in the Gastrointestinal Tract of Goats with LiF Dosimeters After a Single Intake of Simulant Mixed Fission Products, Report F0A-4-4418, The Research Institute of National Defense, Stockholm, Sweden, March 1970.
5. J. S. Cheka, E. M. Robinson, L. Wade, Jr., and W. A. Gramly, The UT–AEC Agricultural Research Laboratory Variable Gamma Dose-Rate Facility, *Health Phys.*, in preparation.
6. S. Brody, *Bioenergetics and Growth*, Reinhold Publishing Coorporation, New York, 1945.
7. M. C. Bell, Flexible Sealed ^{90}Sr–^{90}Y Sources for Large Area Skin Irradiation, *Int. J. Appl. Radiat. Isotop.*, **21**: 42–43 (1970).
8. C. S. Hobbs, S. L. Hansard, and E. R. Barrick, Simplified Methods and Equipment Used in Separation of Urine from Feces Eliminated by Heifers and by Steers, *J. Anim. Sci.*, **9**: 565–579 (1950).
9. L. Wade, Jr., R. F. Hall, L. B. Sasser, and M. C. Bell, Radiation Dose to the Gastrointestinal Tract of Sheep Fed an Insoluble Beta-Emitter, *Health Phys.*, **19**: 57–59 (1970).
10. J. L. West, M. C. Bell, and L. B. Sasser, Pathology of Gastrointestinal-Tract Beta-Radiation Injury, this volume.
11. L. A. George and L. K. Bustad, Comparative Effects of Beta Irradiation of Swine, Sheep, and Rabbit Skin, in *Swine in Biomedical Research*, Proceedings of a Symposium, Richland, Wash., July 19–22, 1965, L. K. Bustad and R. O. McClellan (Eds.), USAEC Report CONF-650708, pp. 491–500, Battelle–Northwest.
12. D. G. Brown, R. A. Reynolds, and D. F. Johnson, Late Effects in Cattle Exposed to Radioactive Fallout, *Amer. J. Vet. Res.*, **27**: 1509–1514 (1966).
13. M. M. Nold, R. L. Hayes, and C. L. Comar, Internal Radiation Dose Measurements in Live Experimental Animals, *Health Phys.*, **4**: 86–100 (1960).

14. D. G. Brown, Clinical Observations on Cattle Exposed to Lethal Doses of Ionizing Radiation, *J. Amer. Vet. Med. Ass.,* **140:** 1051–1055 (1962).
15. M. C. Bell, Livestock and Postattack Recovery, in Proceedings of the Symposium on Postattack Recovery from Nuclear War, Fort Monroe, Va., Nov. 6–9, 1967, Report CONF-671135, pp. 43–58, Office of Civil Defense, Office of Emergency Planning, and National Academy of Sciences–National Research Council, April 1968.
16. A. H. Boerma, A World Agricultural Plan, *Sci. Amer.,* **223:** 54–69 (1970).
17. USDA Consumer and Marketing Service, Livestock Market News, **38:** 260 (Mar. 17, 1970).
18. J. E. Johnson and A. I. Lovaas, Deposition and Retention of Simulated Near-In Fallout by Food Crops and Livestock. Technical Progress Report No. 1, Report AD-695683, Colorado State University, May 1969.
19. S. A. Griffin, Vulnerability of Livestock to Fallout Radiation, in Annual Progress Report, Civil Defense Research Project, March 1968–March 1969, USAEC Report ORNL-4413(Pt. 1), pp. 94–96, October 1969.
20. R. S. Lowrey and M. C. Bell, Whole-Body Irradiation in the Young Pig: Growth, Hematology and Metabolism of ^{45}Ca and ^{89}Sr, *Radiat. Res.,* **23:** 580–593 (1964).
21. R. G. Cragle, J. K. Miller, E. W. Swanson, and D. G. Brown, Lactation and Radionuclide Metabolism Responses of Dairy Cattle to Lethal Doses of Gamma and Neutron Radiation, *J. Dairy Sci.,* **48:** 942–946 (1965).
22. E. W. Swanson, R. G. Cragle, and J. K. Miller, Effects of Irradiation upon Lactation, *J. Dairy Sci.,* **48:** 563–568 (1965).
23. D. K. Wetherbee, Gamma Irradiation of Birds' Eggs and the Radiosensitivity of Birds, Bulletin No. 561, USAEC Report TID-24521, Massachusetts Agricultural Experiment Station, October 1966.
24. UT–AEC Agricultural Research Laboratory, Annual Progress Report, Jan. 1–Dec. 31, 1968, USAEC Report ORO-672, October 1969.
25. B. H. Erickson and R. L. Murphree, Limb Development in Prenatally Irradiated Cattle, Sheep and Swine, *J. Anim. Sci.,* **23:** 1066–1071 (1964).
26. G. A. Malinin (Ed.), *Civil Defense in Rural Regions—A Textbook,* Military Publishing House of the USSR Ministry of Defense, Moscow, 1965. (STS Order No. 5666 for ORNL.)

PATHOLOGY OF GASTROINTESTINAL-TRACT BETA-RADIATION INJURY

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ABSTRACT

Fifty-five wether lambs of mixed breeding and seventeen yearling grade Hereford steers fed ^{90}Y-labeled sand as a fallout simulant developed characteristic lesions, particularly in the upper digestive tract. These changes occurred in selective areas in the stomach compartments. Typically there were large, friable, yellowish, elevated areas of fibrino-necrosis in the rumen sacs; areas of fibrino-necrosis or hemorrhagic necrosis in the reticulum; small hematomas, linear erosions, and focal yellowish necrotic exudate in the omasum; and areas of hemorrhagic necrosis in the abomasum. Healing occurred by scar-tissue formation. Scars in many instances had tags of necrotic exudate and/or superficial erosions months later. Changes in the reticulum and omasum were of appreciably higher incidence and severity in the steers than in sheep. Intestinal lesions were also of increased incidence and severity in steers as compared to sheep. Exposure of animals to beta skin-plaque irradiation in addition to feeding radionuclide did not significantly influence gastrointestinal-tract involvement, but whole-body irradiation exerted a definite additive effect. The conclusion that steers are more sensitive to the effects of the irradiation procedures employed than are sheep appears to be valid.

Livestock grazing in the area of a surface thermonuclear detonation would incur injury and/or death as the result of exposure to external gamma irradiation, skin-surface contamination with fallout beta particles, ingestion of fission products, or as combinations of these exposures. The extent of injury would depend on numerous factors, many of which have been discussed in the light of the possibilities of such an occurrence.[1-3]

Nold, Hayes, and Comar,[4] after feeding soluble ^{90}Y to dogs and goats, concluded that the lower large intestine was the critical organ. These observations were cited in a subsequent report[1] to serve as models for grazing animals. Ekman, Funkqvist, and Greitz[5] found the highest beta concentration in the terminal colon of adult goats treated with a mixture of ^{153}Sm and ^{140}La.

The omasum was the organ severely damaged in the majority of sheep orally treated with soluble ^{144}Ce–^{144}Pr; injury to the rumen was found in only one animal. No changes were observed in the large intestines at levels that were lethal to about 25% of the sheep.[6] Plutonium microspheres in gelatin capsules administered to miniature swine by stomach tube produced macroscopic necrotic and inflammatory areas in the lymphoid tissue at the ileo–cecal junction. Focal microscopic changes were detected throughout the small intestine.[7] Clark reported that insoluble ^{90}Sr administered orally to pigs produced areas of damage in the ileum, cecum, and colon but that the principal lesions occurred in the stomach (see discussion of Ref. 7).

The paucity of information regarding the effects in ruminants resulting from the ingestion of radioactive fallout products and the necessity of these data for arriving at a more realistic evaluation of the results of a nuclear detonation prompted this study.

EXPERIMENTAL PROCEDURE

The experimental design for these studies has been previously described.[9-11] Of the experimental animals, 63 yearling wether lambs of mixed breeding, including 8 untreated controls, and 17 treated yearling Hereford steers were subjected to necropsy. Procedures involving the preparation and feeding of ^{90}Y-labeled sand were previously reported.[9] Skin of the dorsal thoracolumbar region was beta irradiated by the method described by Bell[12] to expose about 8 and 12% of the body surfaces of steers and sheep, respectively. An estimated 57,000 rads was delivered to the exposed skin area in a 3-day period. In animals subjected to bilateral whole-body irradiation, an exposure of 240 R from ^{60}Co sources was delivered at 1 R/min. The number of sheep examined and the treatments were: 38 sheep fed 1.0 to 4.0 mCi of ^{90}Y-labeled sand per kilogram of body weight for 1 to 3 consecutive days; 7 sheep fed ^{90}Y-labeled sand and exposed to skin irradiation; 3 sheep fed ^{90}Y-labeled sand and exposed to whole-body irradiation; and 7 sheep subjected to a combination of the three treatments. Steers were similarly treated: 3 steers fed ^{90}Y-labeled sand at the rate of 2.0 mCi per kilogram of body weight for 3 consecutive days; 3 steers fed ^{90}Y-labeled sand and exposed to skin irradiation; 4 steers fed ^{90}Y-labeled sand and exposed to whole-body irradiation; and 7 steers subjected to the combined treatments.

Most of the animals were examined in extremis or promptly after death. Some animals were destroyed and examined several months posttreatment (PT). The day of postmortem examination indicates the time period between final treatment and examination; e.g., day 2 indicates that the animal was examined 48 hr after the last dose of ^{90}Y. Representative blocks of tissues were fixed in 10% buffered formalin, dehydrated in alcohol, mounted in paraffin, sectioned at 6 μ, and routinely stained with hematoxylin and eosin or special staining procedures if conditions indicated.

RESULTS

General

In both ovine and bovine species, the most extensive pathologic changes occurred in the floor of the caudal half of the ventral ruminal sac (VRS). Frequently involvement of the VRS and posterior ventral blind sac (PVBS) was continuous. Changes in decreasing severity and extent were present in the anterior ventral blind sac (AVBS), the PVBS, and the posterior dorsal blind sac (PDBS) of the rumen. Frequently groups of papillae 2 to 5 cm in diameter in the vicinity of necrotic lesions were "matted" together or coalesced and were dull reddish gray and rather firm. Other individual papillae were enlarged and deep red, and the apexes were shrunken and hard. The posterior wall and/or the floor of the reticulum was principally affected in cattle but was seldom affected in sheep. Omasal alterations were minor and involved the ventral or free aspects of the major laminae, usually adjacent to the reticulo–omasal orifice. In the abomasum the greater curvature of the caudal fundus and adjacent pylorus were the predominant sites of injury. Frequently the involvement extended for variable distances anteriorly between two or more fundic spiral folds. The mucosa was edematous, hyperemic, and frequently studded with petechial and ecchymotic hemorrhages. Spiral folds surrounding ulcers often had sloughed. Subserous hemorrhages and gelatinous infiltration occurred frequently, especially over mucosal lesions. Fibrinous and fibrous adhesions were commonly observed between organs and/or the abdominal floor. The entire thickness of the walls of the rumen, reticulum, and abomasum was affected in moderately severe and severe lesions.

Sheep

The severity of lesions was variable; usually lesions produced were proportional to the amount of radionuclide fed. The usual biologic variation, however, was observed.[2,3,13]

Oral Treatment

No lesions were detected in sheep examined at days 0, 1, and 2. An ovoid, tan, elevated, necrotic plaque (3 by 4 cm) with several polypoidlike nodules around the periphery was observed in the VRS on day 3. Five smaller, soft, fluctuating, tan, polypoidlike nodules were in the floor of the PVBS. Similar ruminal changes were observed on day 5, and a few small hematomas involved two omasal laminae. Similar and somewhat more extensive changes were found in all ruminal compartments on day 6. A small tan nodule was observed in the reticulum. A few superficial erosions and ecchymotic hemorrhages were seen on a few omasal laminae. The abomasal mucosa was hyperemic, with a few lineal hemorrhages on the free borders of a few fundic spiral folds. An area of hemorrhagic necrosis (3 by 4 cm) with a fibrinous exudate was observed in the caudal fundus. Ruminal changes were similar but more extensive by day 7, but the reticulum and omasum were unchanged. A large area of hemorrhagic necrosis involved the abomasum. On day 9 more extensive but similar ruminal involvement and a few yellowish nodules in the reticulum were observed. A few major omasal laminae had superficial linear erosions and a few adherent yellowish nodules. Abomasal changes were somewhat less severe than on day 7.

Similar but less extensive ruminal changes were observed on days 10 and 11. The reticulum and omasum were not affected. The abomasal mucosa and submucosa were markedly edematous and hyperemic with a small area (1 by 1.5 cm) of hemorrhagic necrosis. Ruminal changes on day 13 were similar to those observed on day 9, and there were no alterations in the reticulum and omasum. The abomasal mucosa was slightly hyperemic and edematous.

A Y-shaped, partially healed scar with scattered necrotic tags was observed in the AVBS on day 17. Fibrino-necrotic plaques in the other compartments were detaching at the edges or "rolling up", exposing granular hemorrhagic bases. Similar changes were seen on day 18, but the surface exposed by the detaching, friable, necrotic plaques was pale and smooth.

Similar ruminal changes were observed on day 21. An elliptical area of hemorrhagic necrosis in the abomasum was covered with a mottled, reddish-tan, fibrino-necrotic exudate. There was a moderate amount of sanguineous fluid and of clear, yellowish fluid in the abdominal and thoracic cavities, respectively. The lungs were expanded, heavy, reddish gray in color, and edematous.

Stellate bluish scars with scattered necrotic tags were seen in ruminal compartments on day 57. The caudal fundus and cephalic pylorus of the abomasum over an area measuring 7 by 10 cm were firmly adherent to the abdominal wall by dense fibrous tissue. An ulcer 4 cm in diameter extended almost to the skin. The skin overlying this area was cyanotic and rather firm. Sheep examined on days 72, 298, 307, 344, 365, and 372 had stellate, grayish-white, ruminal and abomasal scars. Several scars were studded with variable-sized superficial erosions.

The mucosa of the proximal duodenum was frequently congested and edematous. Changes in other portions of the intestines were insignificant.

Severe Complications

One sheep developed a ruminal fistula on day 132. A thick-walled, fistulous tract 3.5 cm in length and 1.5 by 2.5 cm in diameter extended from the anterior aspect of the posterior pillar of the VRS to the exterior, emerging about 1.3 cm anterior to the prepuce. The pillar was eroded. A deep ulcer surrounded by dense fibrous tissue was found in the adjacent PVBS. The rumen in this area was firmly adherent to the abdominal wall by fibrous connective tissue.

A soft, fluctuating, epilated, pendulous enlargement (4 by 6.5 cm) anterior and sagittal to the prepuce was observed in a sheep on day 66. The hernial sac contained 5.5 by 6 cm of the caudal abomasal fundus. The abomasum was firmly attached to the hernial ring and a dirty yellow necrotic exudate covered the mucosa of the herniated tissue. A scar (3 cm) extended into the pylorus from the diverticulum. Eversion-type abomasal prolapse developed in three sheep on days 81, 169, and 201. A similar lesion developed on day 171 in a sheep that received combined oral and skin-plaque treatment. Since the caudal fundus was firmly adherent to the hernial ring by dense fibrous tissue, the cephalic pylorus constituted the major part of the prolapsed tissue. The prolapsed tissue was hyperemic, markedly edematous, and studded with superficial necrotic foci.

Oral and Skin-Plaque Treatment

Combined oral and skin-plaque treatment did not appear to influence significantly the extent of stomach changes; however, these animals were examined 171, 176, 315, 350, 439, and 447 days PT. It is of interest to note that the pericardial fluid was increased in these animals. Myocardial atony and dilated, thin-walled ventricles were associated with this finding.

Oral and Whole-Body Treatment

Three sheep exposed to combined oral and whole-body irradiation were examined 2, 15, and 365 days PT. The exudate of the ruminal lesions of the animal examined on day 15 was blood stained. A large area of hemorraghic necrosis involved the abomasum (Fig. 1).

Oral, Whole-Body, and Skin-Plaque Treatment

Ruminal lesions of a sheep examined on day 19 following combined oral, whole-body, and skin-plaque irradiation were not increased in size, but the exudate contained a significant admixture of blood (Fig. 2). Three fistulous tracts originating from ruminal scars were found in a sheep examined on day 58. These tracts were surrounded by dense, reactive, fibrous tissue. Ruminal scars with superficial erosions were present in a sheep examined 419 days PT.

Steers

Oral Treatment

Steers fed ^{90}Y-labeled sand were examined 13, 42, and 59 days PT. The pharyngeal mucosa of one steer was moderately congested and edematous (day 13). Ruminal changes were grossly similar to those in sheep. These changes consisted of elevated, yellowish to yellowish-green, necrotic plaques frequently accompanied by polypoidlike masses of similar composition. Detachment of the friable necrotic exudate at the borders exposed a roughened, hemorrhagic surface (day 42). The necrotic plaques measured up to 12 by 21 cm and involved

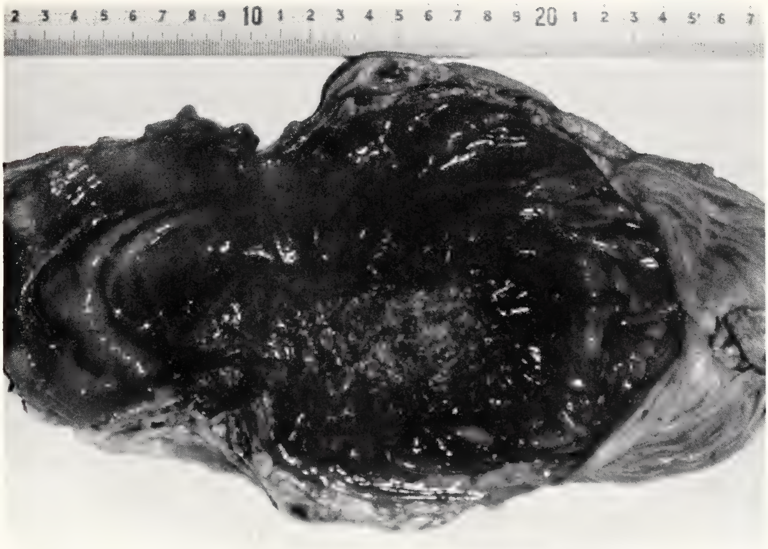

Fig. 1 Abomasum of sheep 41, 15 days after oral and whole-body irradiation. A 5.5- by 11-cm area of hemorrhagic necrosis involving the fundic–pyloric region. Spiral folds in the necrotic area have sloughed. The mucosa and submucosa of the entire organ is hyperemic, edematous, and focally hemorrhagic.

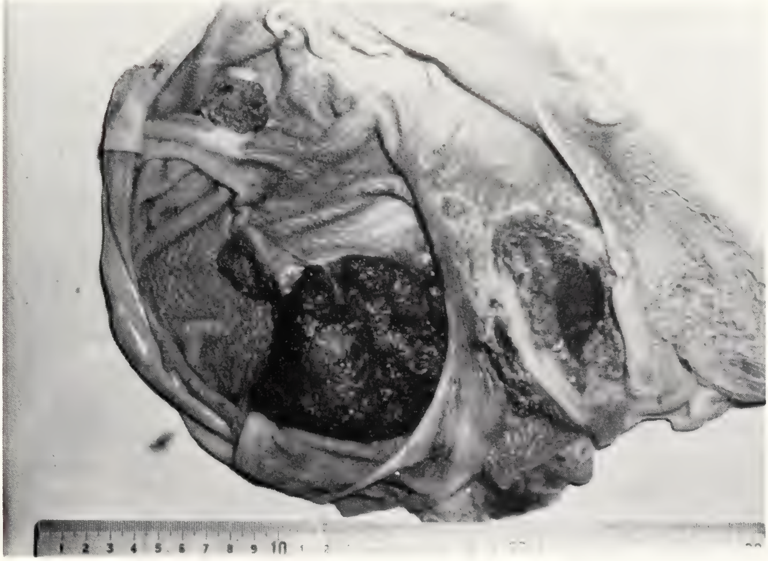

Fig. 2 Rumen and reticulum of sheep 10, 19 days after oral, whole-body, and skin-plaque irradiation. Rumen PDBS (left) fibrino-necrotic plaque; PVBS (lower left) fibrino-hemorrhagic-necrotic plaque; VRS (below) large fibrino-hemorrhagic-necrotic plaque; and AVBS (right) large area of fibrino-necrosis with a large hemorrhagic ulcer in the center. Reticulum (right) is normal.

the entire thickness of the wall. Gelatinous exudation, hemorrhage, and fibrinous or fibrous adhesions to adjacent organs (or less frequently to the abdominal wall) were seen. A tortuous, thick-walled, fistulous tract extended from the postero-medial floor of the VRS to the medial wall of the abomasum (day 59). Variable-sized scars partially covered with necrotic exudate were seen in the ruminal compartments. An area of necrosis (5 by 7 cm) was observed in the reticulum (day 13). A scar (1.5 by 17 cm) studded with small superficial erosions was observed on day 42. Omasal changes were limited to focal congestion of a few major laminae. An area of hemorrhagic necrosis (5 by 8 cm) involved the cephalic pylorus of the abomasum of one steer. The necrotic process had extended into the submucosa of the contiguous fundus (day 13). A tear-shaped scar (5 by 21 cm) with scattered necrotic tags was observed (day 42). The communicating fistulous tract (day 59) from the rumen entered the medial aspect of the terminal abomasal fundus. The spiral folds surrounding the tract had sloughed. A healed scar (3 by 7 cm) was seen in the caudal fundus. The surrounding mucosa was edematous and dirty brownish red in color.

Scattered areas of congestion were observed in the mucosa of the small intestine. A thickened area consisting of numerous nodules up to 1.5 cm in diameter was observed at the ceco—colic junction. The centers of some of the nodules contained yellow, necrotic plugs (day 42). The ileal and colic mucosae (and possibly the submucosa) of one steer were dull grayish red in color and appreciably thickened by transverse ridges (day 59).

An estimated 16 liters of sanguineous ascitic fluid containing yellowish fibrinous aggregates was seen in the steer examined on day 13. Fibrinous tags were adherent to the parietal and visceral peritoneum. Three liters of clear ascitic fluid was present in the steer examined on day 42.

Oral and Skin-Plaque Treatment

Ruminal changes were comparable to those in the previous group (days 20 and 51). Superficial erosions studded the scars of the animal examined on day 300. Depressed stellate scars (2 by 8 cm and 1.5 by 16 cm) were observed in the reticulum (days 20, 51, and 300). Linear erosions and small yellowish nodules of necrotic exudate were observed on some major omasal laminae (day 20). Variable-sized scars (12 to 21 by 2 to 4 cm) were observed in the wall of the greater curvature of the abomasum (days 20 and 51). The scars extended for several centimeters between five laminae (day 20). The surfaces of the scars were partially covered with yellowish-green necrotic exudate. There was a stellate scar (2 by 12 cm) in the caudal fundus and a second scar (1 by 11 cm) in the cephalic pylorus of the abomasum of the steer examined on day 300. Intestinal changes were comparable to those in the previous group.

Oral and Whole-Body Treatment

Elevated, linear and ovoid, dull gray, superficial erosions studded the mucosa of the thoracic portion of the esophagus of steers examined on days 17 and 37.

Ruminal involvement was of increased extent and severity, some plaques measuring 2.5 by 25 by 30 cm. In addition to the thick, yellowish or yellowish-green, friable plaques with polypoid masses (Fig. 3), there were elevated, yellowish areas covered with enlarged, sparse papillae. Some necrotic plaques (up to 5 by 12 by 14 cm) had completely detached and exposed a hemorrhagic granular surface. Necrosis of the reticulum was increased in extent and severity and consisted of large yellow or yellowish-green plaques and areas of hemorrhagic necrosis with sparse necrotic exudate (Fig. 3). Small linear erosions and focal, yellowish, necrotic plaques were observed on a few major omasal laminae in three steers. Abomasal changes were comparable to those in the previous group, the alterations consisting of large areas of hemorrhagic necrosis (Fig. 4) partially covered with yellowish, necrotic exudate (days 12, 15, and 17). The fundic spiral folds were moderately to markedly edematous with scattered ecchymotic hemorrhages. Deep erosions or ulcers occurred between several spiral folds. A scar with a hemorrhagic base was partially covered with cream-colored, necrotic exudate (day 37). The overlying serosa was congested, roughened, and covered with fibrino-hemorrhagic tags.

The duodenal mucosa was congested and edematous with small irregular and linear hemorrhages. Several gray and hemorrhagic nodules 4 to 5 mm in diameter had developed in the mucosae of the lower jejunum, ileum, and the midportion of the cecum (day 37).

Oral, Whole-Body, and Skin-Plaque Treatment

Superficial, grayish-red, linear streaks were observed in the esophageal mucosa (day 12). Changes in the rumen and reticulum were comparable to those in the preceding group. Omasal changes were similar but more extensive, consisting of linear erosions and hemorrhagic necrosis with necrotic exudate. The cavity of the omasum of one steer (day 17) was completely filled with a currant-jelly type of blood clot. There were areas of hemorrhagic necrosis (up to 8 by 28 cm) in the abomasum. The fundic spiral folds were edematous and hyperemic with scattered petechial and ecchymotic hemorrhages. A bluish, depressed, stellate scar (3 by 13 cm) was present in the abomasum of the steer surviving for 52 days.

The mucosa of the small intestine was congested, and in some there were ecchymotic hemorrhages in the wall (days 12, 16, 17, and 18). In one (day 17) several areas of hemorrhage (2 to 7 cm) in the wall with fibrino-hemorrhagic organizations attached to the mucosa were seen. There were fluid blood and blood clots in the lumina. In one steer an ulcer had developed in the mucosa over a large area of subserous hemorrhage (day 18). Cecal changes included scattered ecchymotic hemorrhages in the wall (day 17), solitary ulcers (days 18 and 31), a large ulcer over an area of submucosal hemorrhage (day 18), and an area (4 by 7 cm) with several small ulcers (day 31). The lumina of both the cecum and colon contained fluid blood and blood clots or bloody ingesta. The

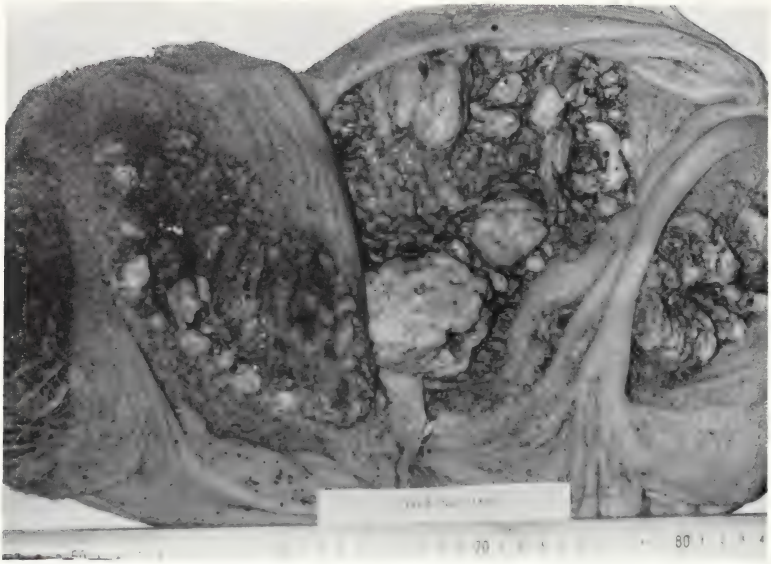

Fig. 3 Rumen and reticulum of steer 185, 17 days after oral and whole-body irradiation. Reticulum (left) with large area of hemorrhagic necrosis. Ruminal compartments (left to right), AVBS, VRS, PVBS, have necrotic plaques with variable-sized polypoidlike masses of exudate.

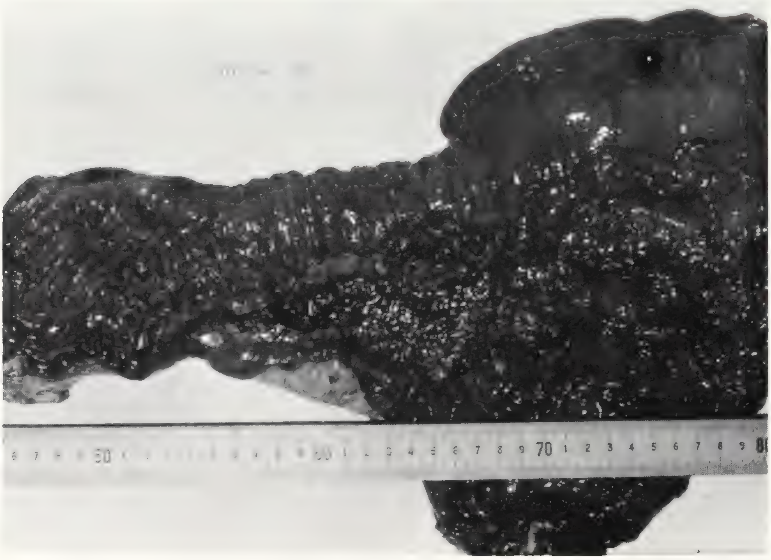

Fig. 4 Abomasum of steer 185, 17 days after oral and whole-body irradiation. A 7- by 19-cm area of hemorrhagic necrosis involving the fundic–pyloric region. Spiral folds in the area are necrotic and have sloughed. The mucosa and submucosa are hyperemic, edematous, and focally hemorrhagic.

mucosa was congested and studded with petechial and ecchymotic hemorrhages with similar hemorrhages being deeper in the wall.

Microscopic Observations

Preliminary microscopic observations are based on the examination of tissues from 12 sheep exposed to oral treatment only.

Days 1 and 2

Foci of "ballooning" or enlarged, rounded, pale staining cells were observed in the mucosae of the rumen and omasal laminae. There were a few foci of superficial necrosis of the abomasal mucosa.

Day 3

Small and larger microcysts formed by rupture of variable numbers of epithelial cells were seen in the ruminal mucosa. Although some cysts involved only the upper layers of cells, in larger cavities the entire epithelial thickness was affected. The cysts contained granular eosinophilic material and cellular debris. The eosinophilic material in many cysts was vacuolated. Larger cysts were covered by the parakeratotic layer only, but the upper border of some smaller cysts was composed of epithelial cells in addition to the parakeratotic layer. The cysts were primarily seen in the apical two-thirds of the affected papillae. The underlying propria was edematous and infiltrated with polymorphonuclear leucocytes (PMN cells). There were numerous areas consisting of groups of enlarged papillae. The lamina propria was edematous and contained strands of fibrin, and the submucosa was moderately edematous. Foci of necrosis, PMN-cell infiltration, and edema were seen in the abomasal mucosa. A slight fibrinocellular exudate covered the necrotic surface.

Day 5

Focal sloughing of groups of necrotic ruminal papillae exposed the submucosa in some areas. Groups of several papillae were distended with plasma and fibrin; this situation created a honeycomb effect within the propria. There were large areas of fibrino-necrosis of the mucosa (Fig. 5). Hemorrhage and large numbers of PMN cells, many degenerating, occurred in the necrotic mass. The upper submucosa was moderately edematous and extensively infiltrated with PMN cells. The blood vessels were dilated, and the walls of some vessels were necrotic. The vascular endothelium was swollen, vacuolated, or hyperchromatic. In some vessels the endothelial cells were not evident. The deeper submucosa and circular muscle layer were slightly to moderately edematous and infiltrated with inflammatory cells.

There were foci of necrosis and sloughing of the omasal mucosa. The submucosa was moderately edematous and infiltrated with PMN cells. There were foci of hemorrhage. Large areas of hemorrhagic necrosis involved the abomasal mucosa. In some areas a thin layer of necrotic epithelium covered a thick layer of hemorrhage which appeared to rest on a thin rim of necrotic

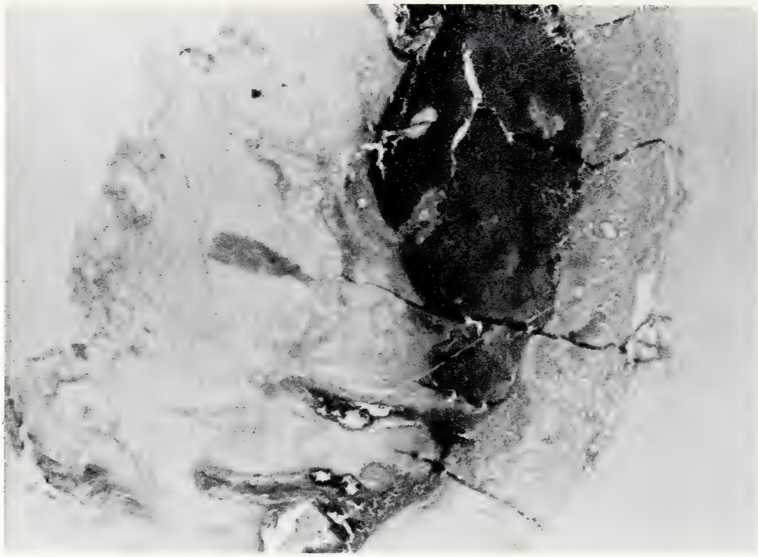

Fig. 5 Rumen of sheep 191, 5 days after oral treatment. Right to left, marked subserous edema. The mucosa is necrotic and covered with a thick fibrinous organization. Remnants of necrotic mucosa on the surface and two necrotic laminae propria (left lower center).

mucosa and the muscularis mucosae. It appeared that rapid and forceful hemorrhage had "lifted" the necrotic mucosa into the lumen. Blood vessels at the base of the mucosa and adjacent glands were dilated. Some of the vessels were characterized by necrotic walls and some by thrombosis. The muscularis mucosae was focally interrupted. The submucosa was markedly thickened by edema and hemorrhage and was extensively infiltrated with PMN cells. Some blood vessels in the upper submucosa had necrotic walls, and some of these vessels contained thrombi. The inner muscle layer bundles were separated by edema.

Day 9

There were large areas of fibrino-necrosis of the ruminal mucosa. Groups of papillae were distended with plasma containing fibrin. The submucosa beneath the large, necrotic, mucosal areas had necrosis and edema and only a few inflammatory cells. Numerous blood vessels in this area were necrotic and thrombotic. In other areas the submucosa was edematous, focally hemorrhagic, and extensively infiltrated with PMN cells. Focal necrosis of the inner muscle layer occurred beneath the more severely affected mucosa and submucosa.

Foci of superficial necrosis and large areas of hemorrhagic necrosis involved the abomasal mucosa (Fig. 6). A large fibrino-hemorrhagic organization was

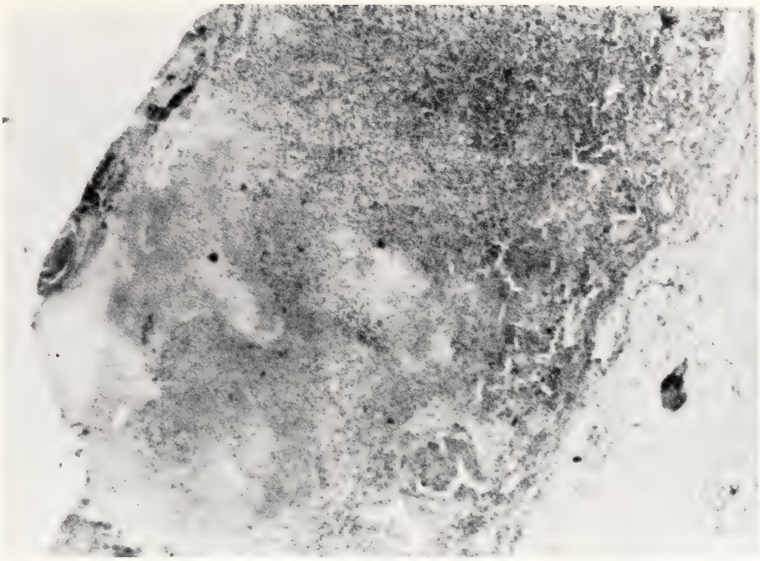

Fig. 6 Abomasum of sheep 175, 9 days after oral treatment. Right to left, extensive submucosal edema and focal hemorrhage. Necrosis and interruption of the muscularis mucosa. Hemorrhagic necrosis of the mucosa with dilated, necrotic, and thrombosed vessels at the base of the mucosa. The surface of the hemorrhagic-necrotic exudate is covered with a thin layer of necrotic mucosa.

attached to the surface in one area. Other changes were similar to those found on day 5.

Day 11

Large areas of fibrino-necrosis of the ruminal mucosa were covered at some sites by necrotic epithelium and the parakeratotic layer. The latter was quite well preserved. Epithelial cells bordering the necrotic areas were enlarged and rounded and the nuclei were pyknotic. Some rete pegs were irregular in shape and of increased length. The underlying propria was edematous and extensively infiltrated with PMN cells. The submucosa was moderately edematous and focally hemorrhagic. There was a moderate infiltration of PMN cells with fewer lymphocytes and mononuclear cells. Collagen fibers in the upper submucosa were anuclear, swollen, and dull red, and some fibers were "frayed." The walls of some blood vessels were necrotic, and some vessels were thrombotic. The abomasal changes were comparable to those observed on day 9.

Days 13 and 18

The changes were similar to those found on day 11.

Day 59

The lining of large areas of the rumen consisted of a mixture of vascular granulation tissue and fibroblasts. The fibroblasts were oriented parallel to a surface that was "ragged" and superficially necrotic. The underlying collagen fibers were swollen. In other areas a layer of dark epithelium with a thickness of two to three cells formed the inner lining. The undulant surface had no papillae. Rete pegs were absent, sparse and short, or sparse, long, and irregular. The edematous submucosa was extensively infiltrated with macrophages. Several blood vessel walls were eccentrically thickened.

Changes in the abomasal mucosa included dilated glands, glandular atrophy, atrophy and glandular degeneration with moderate mononuclear infiltration and slight infiltration of lymphocytes and PMN cells, focal superficial necrosis, necrosis of the entire mucosa, and ulcer formation. A few colonies of large bacterial rods were seen beneath the necrotic mucosa. A large area of the submucosa forming the base of the ulcer was replaced by vascular granulation tissue and fibroblasts. This tissue was moderately infiltrated with macrophages and PMN cells. Coagulation necrosis involved another large area of the submucosa beneath the ulcer. Several dilated, necrotic, and thrombosed vessels were seen in this area. A band of caseous necrosis involved the lower submucosa and a portion of the thin muscle layer. The atrophic muscle layer rested on a thick layer of collagenous fibers and contained islands of granulation tissue and fat. Skin was not present on the sections.

DISCUSSION

Regressive cellular changes and cellular necrosis produced by irradiation are not pathognomonic.[13-15] Similar changes have been produced by a variety of causes.[13] The exact mechanism or mechanisms by which cellular changes are produced by irradiation are not known but are probably multiple.[13-15]

The pharyngeal mucosa and submucosa were congested and edematous, and the esophageal mucosa of a few steers had linear and ovoid erosions. It is probable that these changes occurred during regurgitation of ruminal fluids rather than as a consequence of ingestion of feed containing the radionuclide.

Yttrium-90-labeled sand ingested by sheep and cattle collects in rather specific ruminal and abomasal sites and produces characteristic pathologic lesions. Sand particles lodge between ruminal papillae in these areas and appear to be indefinitely retained by the ensuing inflammatory and necrotic exudate. Ruminal contractions and compartmentalization by the pillars probably are important in determining the areas where radioactivity will be concentrated. In a few early lesions, focal accumulation of plasma beneath and within the mucosa resulted in dome-shaped, yellowish elevations sparsely covered with enlarged papillae. Later, necrosis of the mucosa, increased vascular damage, extensive effusion of plasma, and extensive inflammatory cell infiltration produced the characteristic large fibrino-necrotic plaques or masses observed in sheep. Probably the grossly similar lesions seen in cattle would be comparable microscopically. Detachment of the necrotic masses at the borders exposed a hemorrhagic, granular base or a smooth, pale surface, the appearance depending upon the age of the lesion. A pale, depressed, stellate scar was apparent on detachment of the exudate. Several months after treatment necrotic tags and superficial erosions were seen on the surfaces of numerous scars.

The reticulum was mildly affected in a few sheep. In contrast, necrotic plaques or areas of hemorrhagic necrosis were seen in the reticulum of a significant number of steers. We have no explanation for this species difference.

In general, minor lesions only were seen in the omasum of a few sheep. In steers the changes were of appreciably greater incidence and severity. The omasum of one steer was filled with a currant-jelly blood clot. An area of hemorrhagic necrosis between two laminae had apparently eroded into a large blood vessel.

Characteristically injury occurred at the fundic–pyloric region on the greater curvature of the abomasum. This selective location is probably due to gravitational forces, the sand particles settling in the lowest area of the organ. Several variable-sized extensive areas of hemorrhagic necrosis developed in this area. Some lesions were covered in part with a thick fibrino-necrotic exudate.

Anorexia (in the absence of more-severe complications) following treatment for variable periods resulted in appreciable weight loss. Ruminal fistula, abomasal hernia, and eversion-type abomasal prolapse occurred in six sheep. Another sheep probably would have developed an abomasal fistula if it had survived. Fibrinous and fibrous adhesions of organ to organ and/or to the abdominal floor occurred frequently in sheep. Similar adhesions between organs were frequently seen in steers. Only two steers developed ruminal adhesions to the abdominal floor. In one steer a long, tortuous, communicating fistulous tract extended from the rumen to the abomasum. The cause of this development is obscure. Fibrous adhesions of organ to organ or to the abdominal floor would interfere with normal function and conceivably could result in strangulation. Transportation and other stress-producing experiences may cause separation of adhesions and subsequent peritonitis.[16]

The absence of significant intestinal lesions in sheep was unexpected. Intestinal lesions found only in orally treated steers were not severe. The ileal, cecal, and colic mucosae (and possibly submucosae) of the intestine of one steer were appreciably thickened by transverse ridges. This change was not believed to be associated with irradiation, but microscopic examination has not been completed.

Whole-body irradiation superimposed on oral treatment appeared to increase the extent and severity of gastrointestinal changes.

Focal microcyst formation and foci of epithelial necrosis were early ruminal mucosal changes. Microcysts were probably the result of cellular imbibition of fluid and subsequent rupture of the cells. The cysts were frequently multiple on papillae and involved the apical portions of the affected papillae. The underlying lamina propria was edematous and infiltrated with numerous PMN cells. Microcysts, which are not an unusual ruminal mucosal change in sheep, apparently occur as a result of altered physiology.[8] These cysts are not associated with inflammation of the lamina propria. Focal effusions of plasma into the mucosa caused marked swelling of groups of papillae. The epithelium of these papillae was degenerative or focally necrotic. The propria was distended with proteinaceous fluid and fibrin; this distention created a honeycomblike effect.

In more advanced lesions large areas of fibrino-necrosis involved the mucosa. This exudate consisted of necrotic mucosa, fibrin, and extensive PMN-cell infiltration. In some areas the exudate had sloughed and exposed a congested, ragged submucosa. The submucosa was edematous, focally hemorrhagic, and extensively infiltrated with PMN cells. The blood vessels were dilated. Many were necrotic and several thrombosed. The necrotizing reaction extended to the serosa in more severely affected areas. The inner surface of a ruminal scar was formed by granulation tissue and fibroblasts or a thin (2- to 3-cell thickness) layer of hyperchromatic epithelium with no or with scattered, short rete pegs. The submucosa was edematous and extensively infiltrated with macrophages.

Minor changes of focal necrosis of the omasal mucosa with edema and cellular infiltration of the submucosa were seen.

Hemorrhagic necrosis was the characteristic change seen in the abomasal mucosa. The submucosa was markedly edematous and focally hemorrhagic. Many blood vessels were necrotic and thrombosed. In one animal a chronic ulcer had developed. The underlying submucosa was replaced in one area by vascular granulation tissue and fibroblasts, which were infiltrated with PMN and mononuclear cells. A large area of coagulation necrosis involved an adjacent area of the submucosa beneath the ulcer, indicating concomitant repair and continuation of an acute reaction.

Intestinal changes in sheep were minimal. Comparable treatment of cattle induced significant lesions. Bacterial invasion of tissue was observed in only a few animals. The conclusion that sheep are less sensitive to the radiation procedures employed than are cattle appears to be justified.

ACKNOWLEDGMENTS

The UT–AEC Agricultural Research Laboratory is operated by the Tennessee Agricultural Experiment Station for the U. S. Atomic Energy Commission under Contract AT-40-1-GEN-242.

This work was supported by funds from the U. S. Office of Civil Defense and is published with the permission of the Dean of the University of Tennessee Agricultural Experiment Station, Knoxville.

REFERENCES

1. National Academy of Sciences–National Research Council, Damage to Livestock from Radioactive Fallout in Event of Nuclear War, Publication 1078, Washington, D. C., Dec. 20, 1963.

2. J. H. Rust, Report of the National Academy of Sciences Subcommittee for the Assessment of Damage to Livestock from Radioactive Fallout, *J. Amer. Vet. Med. Ass.*, **140**: 231–235 (1962).
3. H. A. Smith and T. C. Jones, *Veterinary Pathology*, pp. 658–673, Lea & Febiger, Philadelphia, 1966.
4. M. M. Nold, R. L. Hayes, and C. L. Comar, Internal Radiation Dose Measurements in Live Animals, *Health Phys.*, **4**: 86-100 (1960).
5. L. Ekman, B. Funkqvist, and U. Greitz, Beta- and Gamma-Dose Measurements in the Gastrointestinal Tract of Goats with LiF Dosimeters After a Single Intake of Simulant Mixed Fission Products, Report FAO-4-4418, The Research Institute of National Defense, Stockholm, Sweden, March 1970.
6. M. C. Bell, Airborne Radionuclides and Animals, in *Agriculture and the Quality of Our Environment*, N. C. Brady (Ed.), pp. 77-90, Symposium No. 85, American Association for the Advancement of Science, Washington, D. C., 1967.
7. V. H. Smith, H. A. Ragan, B. J. McClanahan, J. L. Beamer, and J. L. Palotay, The Passage Time of Plutonium Oxide in Pigs, in *Gastrointestinal Radiation Injury*, M. F. Sullivan (Ed.), Report of a Symposium held at Richland, Wash., Sept. 25–28, 1966, Report CONF-660917, pp. 518–523, Exerpta Medica Foundation, New York, 1968.
8. Alfred Trautmann and Josef Fiebiger, *Fundamentals of the Histology of Domestic Animals*, pp. 182–183, Translated and revised from the eighth and ninth German editions, 1949, by Robert E. Habel and Ernst Biberstein, Comstock Publishing Associates, Ithaca, N. Y., 1952.
9. M. C. Bell, L. B. Sasser, J. L. West, and L. Wade, Jr., Effects of Feeding ^{90}Y-Labeled Fallout Simulant to Sheep, *Radiat. Res.*, **43**: 71–82 (1970).
10. L. B. Sasser, M. C. Bell, and J. L. West, Simulated-Fallout-Radiation Effects on Sheep, this volume.
11. M. C. Bell, L. B. Sasser, and J. L. West, Simulated-Fallout-Radiation Effects on Livestock, this volume.
12. M. C. Bell, Flexible Sealed ^{90}Sr–^{90}Y Sources for Large Area Skin Irradiation, *Int. J. Appl. Radiat. Isotop.*, **21**: 42–43 (1970).
13. W. A. D. Anderson, *Pathology*, Vol. I., pp. 166–188, The C. V. Mosby Company, St. Louis, Mo., 1966.
14. A. R. Moritz and F. W. Henriques, Jr., Effects of Beta Rays on the Skin as a Function of the Energy, Intensity, and Duration of Radiation. II. Animal Experiments, *Lab. Invest.*, **1**: 167–185, 1952.
15. Philip Rubin and G. W. Casarett, *Clinical Radiation Pathology*, Vol. I, pp. 1–37, W. B. Saunders Company, Philadelphia, 1968.
16. K. Nieberle and P. Cohrs, *Textbook of the Special Pathological Anatomy of Domestic Animals*, revised by Paul Cohrs, translated by R. Crawford, pp. 364–369, Pergamon Press, Inc., London, 1967.

RESPONSES OF LARGE ANIMALS TO RADIATION INJURY

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ABSTRACT

Recent data pertaining to the relations between dose rate and lethality in sheep exposed to dose rates ranging from high (hundreds of roentgens per hour) to low (less than 1 R/hr) are incorporated in a review of the field. It is concluded that even within the high dose-rate range there is a significant inverse relation between $LD_{50/60}$ and dose rate and that discernible radiation injury does accrue even at dose rates less than 1 R/hr. The chronology of lethality and hematologic changes in sheep during continuous exposure to death at dose rates of 3.74 and 1.96 R/hr are compared with those observed during terminated exposure at 0.84 R/hr. At 1.96 R/hr there is no indication of reduction in survival time by overirradiation, whereas at 3.97 R/hr there is a marked compression in the range of survival times. Chronology and extent of changes in circulating leukocyte counts vary appreciably with dose rate during protracted exposure.

In April 1968, at the symposium on dose rate in mammalian radiation biology,[1] Dr. Norbert Page gave an overview of the effects of dose protraction on radiation lethality in large animals. His summary, together with some other papers presented at that symposium, furnished an excellent statement of the state of the art at that time. In his summary of the entire symposium, Edward Alpen pointed up the importance of describing the effects of variation in dose rate in considering recovery processes and the untenability of the view that a single unique recovery "constant" exists, even for a given species.

My objective is to update the information presented at the 1968 symposium, with particular reference to the sheep. This species is of major interest to this present symposium because the sheep is an economically important domestic animal resource and because it is the large animal that has been most systematically studied with respect to the relations between dose rate and response to radiation.

The information presented comes principally from the most recent technical reports of the Naval Radiological Defense Laboratory (NRDL) program in large animal radiobiology, published during the last months of that laboratory's existence, and from the initial studies under the Office of Civil Defense (OCD) program now located at the Stanford Research Institute (SRI).

Two specific areas are considered: (1) the relation between dose rate and the $LD_{50/60}$ as measured in the terminated type of exposure to a predetermined dose and (2) the relation between dose rate and mortality and hematological responses during continuous exposure to death.

$LD_{50/60}$ AS A FUNCTION OF DOSE RATE

The $LD_{50/60}$ for animals exposed to high dose rates is of interest from two standpoints: Lethality does appear to vary with dose rate even within the range of high dose rates usually described as "acute," and the response to high-dose-rate exposure is used as the standard against which responses to low-dose-rate exposure are compared. For example, one standard way of comparing recovery after, or even during, a low-dose-rate exposure is to compare the $LD_{50/60}$ at a high dose rate in animals previously exposed at the low dose rate with that of previously unexposed, comparable animals. The difference between the two $LD_{50/60}$'s is considered to represent the residual injury remaining from the initial low-dose-rate exposure, and the difference subtracted from the dose given at the low dose rate represents the amount of recovery that has occurred.

Figure 1 summarizes the available information on $LD_{50/60}$ in sheep (California-bred wethers) exposed to dose rates ranging from 30 to 660 R/hr (midline air). All exposures were bilateral (1 MVp X ray) or quadrilateral (^{60}Co), and the two types of radiation sources have been shown to have similar depth-dose characteristics.[2] The data from Refs. 2, 3, and 7 were included in Page's 1969 presentation. Since that time there have been five more determinations of the $LD_{50/60}$ at dose rates in excess of 30 R/hr—two at SRI, two at NRDL,[4,5] and one at the Air Force Weapons Laboratory.[6] The composite of the data of Hanks et al. reported in 1966 and the data of some additional groups reported in 1969 by Taylor et al.[3] changed the original estimate of 252 R to 258 R. One can question whether the 30 R/hr value of Page et al. is a part of the high-dose-rate continuum. It is included here because the exposures took less than a day and because its fit with the protracted dose-rate $LD_{50/60}$ data to be considered is even less apparent. When plotted on a graph, these nine data points appear to be adequately fitted by a linear regression (correlation coefficient, −0.82) expressed by

$$Y = 356 - 0.156\ X \qquad (1)$$

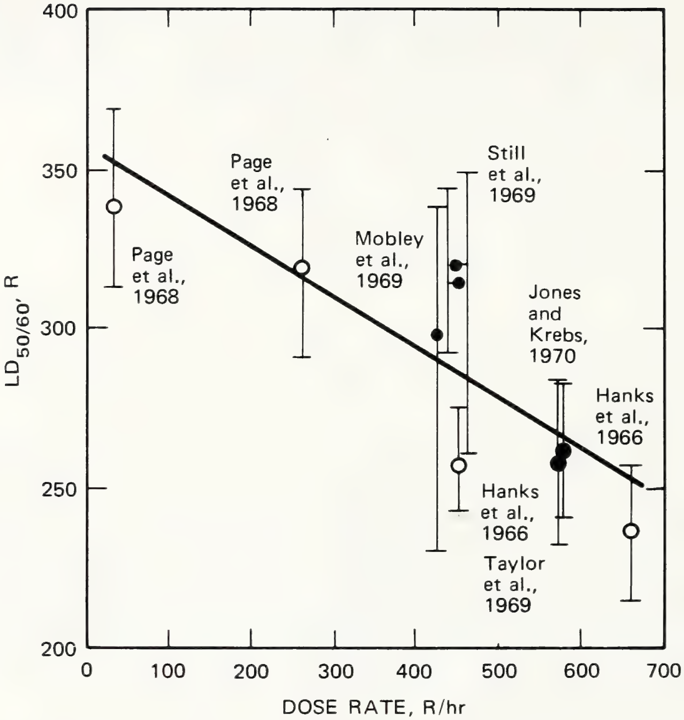

Fig. 1 Relation between $LD_{50/60}$ (midline air) and dose rate in sheep exposed at a high dose rate.

where Y is the $LD_{50/60}$ in roentgens and X is the dose rate in roentgens per hour. Since the 95% confidence interval of the slope (0.093) is less than the computed slope itself (0.156), there is a significant variation of $LD_{50/60}$ with variation in dose rate. Thus, even at dose rates in the so-called acute range, it appears that we should specify the dose rate precisely when describing the $LD_{50/60}$, and, in using acute dose-rate responses to evaluate injury accumulation and recovery at protracted dose rates, we should take into account this variation.

Figure 2 summarizes the presently available $LD_{50/60}$ information for protracted dose rates where the exposure time is of the order of days or weeks. Results of the work by Jones and Krebs, which is currently in progress at SRI, are not sufficient to provide any reliable estimate of the confidence limits for the computed $LD_{50/60}$ at 0.84 R/hr, since there were only three deaths among the five groups of 12 animals exposed. The computed $LD_{50/60}$ of 1084 R is based on one death after exposure at 777 R, one at 837 R, and two at 897 R (the highest dose tested). Evaluation of the characteristics of the relation between dose rate and $LD_{50/60}$ from about 4 R/hr on down appears to be unwarranted until further information is acquired. That there is a tremendous

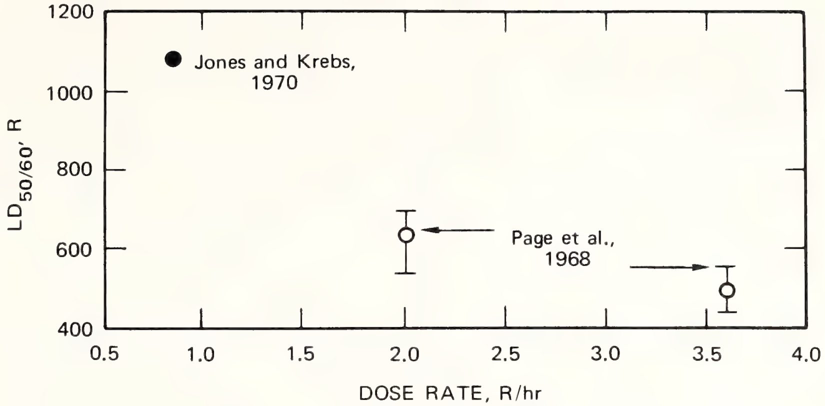

Fig. 2 **Relation between $LD_{50/60}$ (midline air) and dose rate in sheep exposed at a low dose rate to ^{60}Co gamma radiation.**

change in $LD_{50/60}$ as compared with acute dose rates is, of course, quite apparent. It may even be that below some dose rates (presumably less than 1 R/hr) the conventional statistics relating exposure dose and mortality do not apply.

CONTINUOUS EXPOSURE TO DEATH

Figure 3 summarizes lethality and exposure data for the two relatively recent studies of lethality and hematologic changes during continuous (23 hr/day) protracted exposure to death. The first of these was done by Still et al.[8] at NRDL, and the second was done at SRI. At 1.96 R/hr the first death occurred on the 25th day of exposure, the median survival time was 42.5 days, and the last animal died on day 60. Deaths were spread out more or less uniformly throughout the period from days 25 to 60. At 3.79 R/hr, however, there was a marked difference in the lethality pattern. The first death occurred slightly earlier, on day 22, and all the remaining animals died within the next 6 days, the median survival time being 24.5 days. With continuous exposure to death, we are always faced with the concept of irradiation after accrual of a dose lethal to the individual animal. From Fig. 3 it appears that the effect of this so-called "wasted radiation" is a function of the dose rate. At 3.79 R/hr, further exposure after accrual of a potentially lethal dose results in a compression of survival time. It is as though at this dose rate there are no "low-lethal" doses, and animals die with survival times similar to those observed after doses in the high-lethal range for acute exposure. For example, the work of Page et al.[7] indicates that the $LD_{50/60}$ for terminated exposure at 3.6 R/hr is 495 R. In continuous exposure at 3.79 R/hr, this dose was accrued in 5.7 days. Subtracting this from the mean survival time of 24.7 days gives a survival time after accrual of an $LD_{50/60}$ of

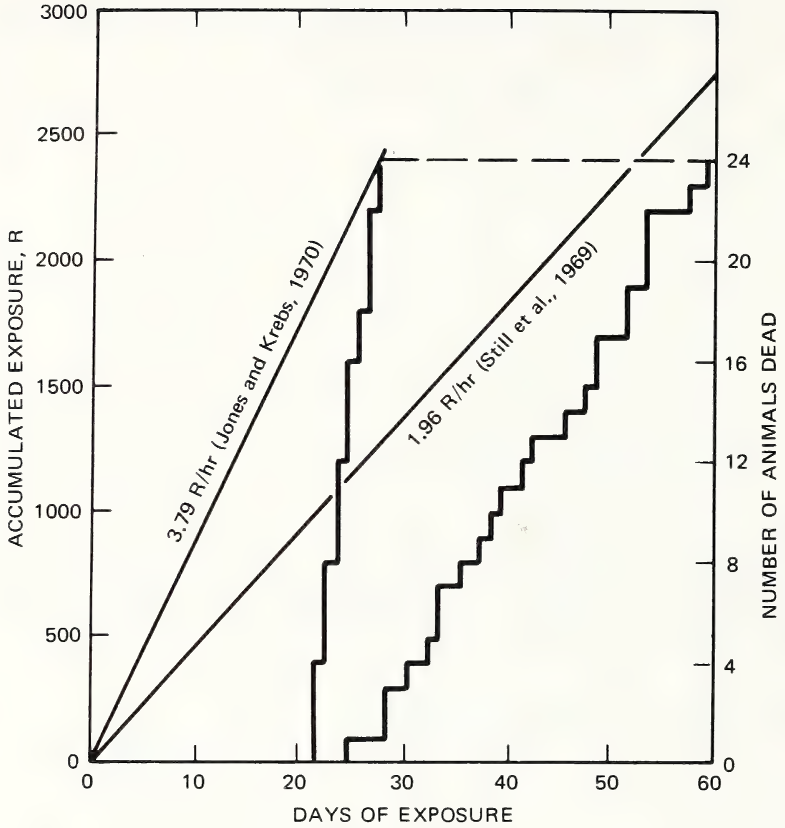

Fig. 3 Cumulative mortality and dose in sheep exposed continuously (23 hr/day) until death at 1.96 or 3.79 R/hr (midline air). Values for 1.96 R/hr are estimated from the data of Still et al.

19 days. This is approximately the value that is typical of survival time when the exposure is near the $LD_{50/60}$ for dose rates of the order of 450 to 600 R/hr. In continuous exposure at 1.96 R/hr, however, there is no discernible compression of the range of survival times. Again, Page et al.[7] found that the $LD_{50/60}$ for 2.0 R/hr (terminated exposure) is 637 R. At 1.96 R/hr this dose is accrued in 14.1 days. Subtracting this value from the mean survival time of 42.9 gives a mean survival time after accrual of an $LD_{50/60}$ of 28.8 days. This is somewhat in excess of the expected mean survival time with exposure at a high dose rate. Thus, although the pattern of lethality with exposure at about 4 R/hr bears some analogy to that seen in acute-dose-rate exposure, survival times at about 2 R/hr present a different pattern.

In our continuous-exposure study at 3.79 R/hr, we took weekly blood samples of all animals beginning on day 9. In our terminated-exposure study at

0.84 R/hr, we took weekly samples during exposure from the highest dose group beginning on day 6. These data are summarized in Figs. 4 to 7, together with weekly values beginning with the seventh day of exposure estimated from the graphs of Still et al.[8] for their continuous 1.96 R/hr study. In considering these data, we should remember that at 1.96 and 3.79 R/hr animals were dying during the period under examination but at 0.84 R/hr there were no deaths during exposure (the two animals of this group which ultimately died survived 22 and 39 days after the last blood sample taken during exposure).

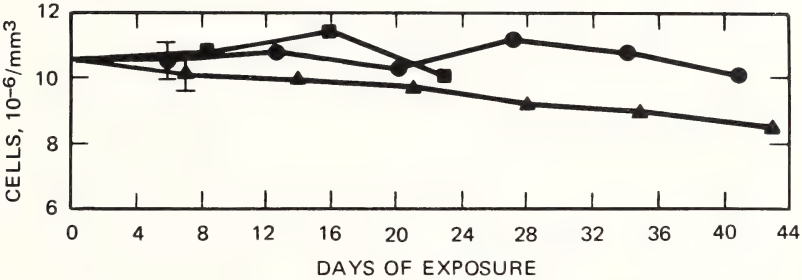

Fig. 4 Values for circulating erythrocytes in sheep during protracted exposure to ^{60}Co gamma radiation. Values for 1.96 R/hr are estimated from the data of Still et al. ●, 0.84 R/hr; ▲, 1.96 R/hr; ■, 3.79 R/hr.

The erythrocyte data for the three studies are shown in Fig. 4. For all three dose rates, there was little appreciable change in red cell count during the first three weeks of exposure. At 3.79 R/hr there was a slight decrease at the fourth week, when half the animals had already died. At 1.96 R/hr this decrease continued during the next three weeks. The red cell count was slightly depressed during the last week of exposure at 0.84 R/hr. Apparently at 3.79 R/hr lethality occurs before the peripheral red cell count responds to depressed erythroid activity in the bone marrow, whereas at 0.84 R/hr the injury accrual rate is too slow to be reflected in the peripheral circulation during the 6 weeks of exposure (red cell counts in this group do show a decrease beginning in the third week after the termination of exposure). Exposure at 1.96 R/hr appears to result in the proper combination of an injury accrual rate high enough and a survival time long enough for depressed red cell counts to be observed.

Total peripheral leukocyte counts are summarized in Fig. 5. Here the pattern among the three studies shows a distinct dose-rate effect. At all three dose rates, there was a definite decrease in total leukocyte count by the first observation after the beginning of exposure, the magnitude of depression being directly related to the dose rate. This initial decrease was followed by a small additional depression at the second observation a week later in all three groups. At the two higher dose rates, there was a further depression in leukocyte count, terminal values being of the order of 13% of the preirradiation level. At 0.84 R/hr the

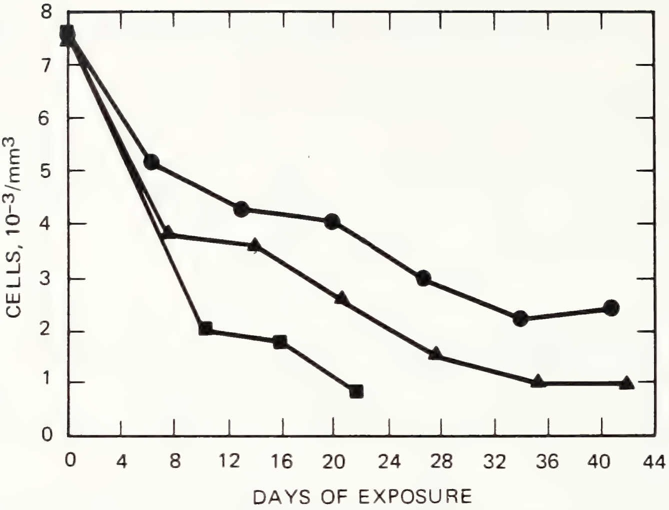

Fig. 5 Values for circulating total leukocytes in sheep during protracted exposure to ^{60}Co gamma radiation. Values for 1.96 R/hr are estimated from the data of Still et al. ●, 0.84 R/hr; ▲, 1.96 R/hr; ■, 3.79 R/hr.

second depression in total leukocytes occurred later, and the final values during exposure were about twice those observed at 1.96 and 3.79 R/hr.

Obviously, changes in total leukocyte counts represent the summation of changes in the myeloid and lymphoid leukocytes. In our work we differentiate the leukocytes only on the basis of whether they are granulocytic or mononuclear cells. For sheep about 85% of granulocytic cells are neutrophils, and 90% of mononuclear cells are lymphocytes. When we examine the changes in these two categories of leukocytes, we find that the dose-rate dependency described for total leukocytes is still there but that there are differences for the two cell categories.

The values for mononuclear leukocytes are summarized in Fig. 6. At either 3.79 or 1.96 R/hr, there was a sharp decline in cell count by about the end of the first week of exposure. This initial depression was complete at 3.79 R/hr, in the sense that the level reached was about 15% of the preirradiation level, but at 1.96 R/hr values about 15% of preirradiation levels were reached after about 3 weeks of exposure. At 0.84 R/hr, values during exposure never declined below about 25% of the preirradiation level, and this range of values was reached after about 3 weeks of exposure. With respect to mononuclear leukocytes, then, there appears to be a fairly discrete dose-rate dependency with respect to the extent of depression and the time of minimum values.

It has been noted before that changes in circulating lymphocytes following whole-body irradiation initially reflect primarily the high radiosensitivity (and consequent death) of circulating lymphocytes and then reflect the decreased

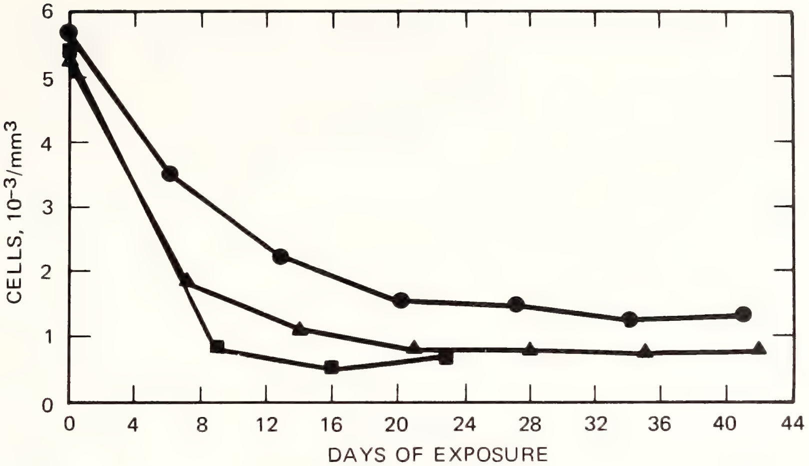

Fig. 6 Values for circulating mononuclear leukocytes in sheep during protracted exposure to ^{60}Co gamma radiation. Values for 1.96 R/hr are estimated from the data of Still et al. ●, 0.84 R/hr; ▲, 1.96 R/hr; ■, 3.79 R/hr.

output of the radiosensitive stem cell system of the bone marrow. Although originally derived for acute irradiation in small animals, this rationale appears to describe satisfactorily the changes in mononuclear leukocytes of the sheep discussed here.

The data for granulocytes are shown in Fig. 7. At 3.79 R/hr there was about a 50% depression by the end of the first week of exposure, no further decrease during the next week, and then a final depression to near-zero values during the next week in the animals surviving long enough to be assayed. At each of the two lower dose rates there was a slight depression in granulocytic cell count after about a week of exposure, then a slight rise during the next week. At 0.84 R/hr this "rebound" persisted for another week. After the apparent abortive rise,

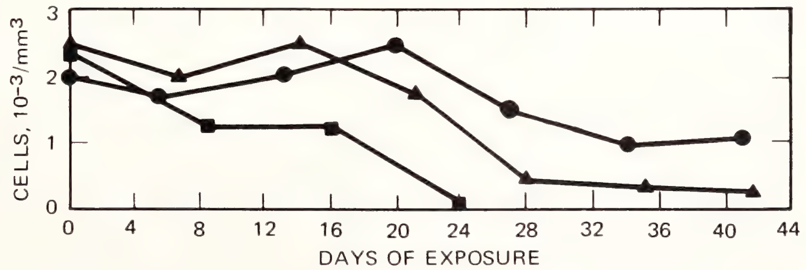

Fig. 7 Values for circulating granulocytes in sheep during protracted exposure to ^{60}Co gamma radiation. Values for 1.96 R/hr are estimated from the data of Still et al. ●, 0.84 R/hr; ▲, 1.96 R/hr; ■, 3.79 R/hr.

granulocytic cell count decreased over the next 2 weeks at both of the lower dose rates. At 1.96 R/hr, values less than 20% of preirradiation levels were observed during the final 3 weeks of observation. At 0.84 R/hr the maximum depression was only to about 50% of the preirradiation value.

In addition to higher radioresistance of circulating granulocytes, as compared with lymphocytes, there is also a considerable reserve of neutrophils available for release into the circulation. For example, Page et al.[9] recently reported that in unirradiated sheep circulating granulocytic cells increase over 300% within a day of injection of endotoxin. As noted by Still et al.,[8] these two factors could account for the chronologic delay in the decrease in circulating granulocytic cells of the sheep during continuous irradiation at doses of 1.96 or 0.84 R/hr. Still et al.[8] also noted that the major point to be made from studies of continuous chronic exposure of large animals was that, unlike the rat, large animals appear unable to adapt to low-level whole-body gamma irradiation. This conclusion obviously appears valid at dose rates of 1.96 R/hr and up. As for lower dose rates, although there was no discernible change in the granulocytic cell count during the last 2 weeks of exposure at 0.84 R/hr (Fig. 7), further decreases have been observed during postexposure observation of these animals (now in progress). This finding, together with the fact that some deaths did occur after exposure, indicate that, although the sheep may be capable of some transient adaptation during protracted exposure, injury does accrue even at a dose rate below 1 R/hr.

REFERENCES

1. D. G. Brown, R. G. Cragle, and T. R. Noonan (Eds.), Proceedings of a Symposium and Dose Rate in Mammalian Radiation Biology, Apr. 29–May 1, 1968, Oak Ridge, Tenn., USAEC Report CONF-680410, UT–AEC Agricultural Research Laboratory, July 12, 1968.
2. G. E. Hanks, N. P. Page, E. J. Ainsworth, G. F. Leong, C. K. Menkes, and E. L. Alpen, Acute Mortality and Recovery Studies in Sheep Irradiated with Cobalt-60 Gamma Rays or 1 MVP X-Rays, *Radiat. Res.*, **27**: 397–405 (1966).
3. J. F. Taylor, E. J. Ainsworth, N. P. Page, and G. F. Leong, Influence of Exposure Aspect on Radiation Lethality in Sheep, Report NRDL-TR-69-15, Naval Radiological Defense Laboratory, Mar. 24, 1969.
4. E. T. Still, J. F. Taylor, G. F. Leong, and E. J. Ainsworth, Mortality of Sheep Subjected to Acute and Subsequent Protracted Irradiation, Report NRDL-TR-69-32, Naval Radiological Defense Laboratory, June 9, 1969.
5. E. T. Still, J. F. Taylor, G. F. Leong, and E. J. Ainsworth, The Influence of the Amount of Initial Radiation Exposure on the Recovery Pattern in Sheep, Report NRDL-TR-69-97, Naval Radiological Defense Laboratory, July 15, 1969.
6. T. S. Mobley, E. T. Still, W. Rush, J. F. Taylor, R. L. Persing, and T. C. DeFeo, Interlaboratory Comparison of Mortality in Sheep Exposed to ^{60}Cobalt Radiation, Report AFWL-TR-69-48, Air Force Weapons Laboratory, Air Force Systems Command, Kirtland Air Force Base, October 1969.
7. N. P. Page, E. J. Ainsworth, and G. F. Leong, The Relationship of Exposure Rate and Exposure Time to Radiation Injury in Sheep, *Radiat. Res.*, **33**: 94–106 (1968).

8. E. T. Still, J. F. Taylor, G. F. Leong, and E. J. Ainsworth, Survival Time and Hematological Responses in Sheep Subjected to Continuous ^{60}Cobalt Gamma Irradiation, Report NRDL-TR-69-28, Naval Radiological Defense Laboratory, May 9, 1969.
9. N. P. Page, E. J. Ainsworth, and J. F. Taylor, Residual Injury of the Hematopoietic System of X-irradiated Sheep, NRDL-TR Report, Naval Radiological Defense Laboratory, July 15, 1969.

EFFECTS OF EXPOSURE TIME AND RATE ON THE SURVIVAL AND YIELD OF LETTUCE, BARLEY, AND WHEAT

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ABSTRACT

Experiments were conducted to compare the effects of ^{137}Cs gamma radiation given as either 1-, 4-, 8-, or 16-hr treatments at constant rates (CR) with 36-hr fallout-decay-simulation (FDS) or with buildup (Bu) and fallout-decay-simulation (Bu + FDS) treatments with variable exposure rates. Seedlings of lettuce were given Bu + FDS, FDS, and 1-, 4-, 8-, and 16-hr CR treatments. Barley and wheat seedlings were given FDS and 8- and 16-hr CR treatments. Following irradiation the lettuce plants were transplanted to the field, barley to the greenhouse, and wheat to a growth chamber. The criteria of effect used were survival and yield. Young barley seedlings were given a total exposure of 1600 R at 32 different rates ranging from 60 to 4800 R/hr. The first leaf of each seedling was measured after 8 days of growth.

For equal total exposures, FDS treatments were more effective than 16-hr CR treatments in reducing survival and yield of all three crops. The ratio of 16-hr CR to FDS at LD_{50} was 1.43 for lettuce, 1.23 for barley, and 1.37 for wheat. For yield the FDS was more effective only at exposures above the LD_{50}. Lettuce survival increased with exposure time between 1 and 16 hr, but this was a linear increase only after 4 hr. Barley seedling height decreased as the exposure rate increased from 60 to about 1000 R/hr. Further increases in exposure rate above 1000 R/hr had no further effect on seedling height. The greater effectiveness of the high exposure rates observed in these experiments substantiates our conclusion that the increased effect of an FDS treatment compared with a 16-hr CR treatment is attributable to the high initial exposure rates of FDS.

Similar results for survival and yield reduction for the 8-hr CR and the FDS treatments were observed. Hence investigators lacking the facilities to simulate fallout decay could use an 8-hr CR treatment to approximate the effects of simulated-fallout-decay treatments.

For equal total exposures of gamma radiation, a treatment simulating fallout decay has been reported[1-3] to be more effective in reducing survival and yield of crop plants than are prolonged constant-exposure-rate treatments. The greater effectiveness of the fallout-decay-simulation (FDS) treatment is thought to be due to the very high exposure rates encountered initially.[1-3] Thus study of the effects of a given amount of fallout or simulated fallout radiation seems to become basically a problem of the effect of variations in exposure rate. This paper presents some of our most recent data on the effects of the gamma component of simulated fallout on crop plants and additional data showing how variations in exposure rate can affect a plant's response to radiation. These data give support to the conclusion that high exposure rates are the basis for the greater effectiveness of the fallout-decay treatments. The plants used in this study were lettuce, barley, and wheat.

MATERIALS AND METHODS

Facilities and Treatment Procedure

The theory and facilities used to simulate fallout decay have been previously described in detail.[1] Basically, a series of stainless-steel shields are lowered over a 12,000-Ci ^{137}Cs source at predetermined times to simulate exposure to fallout radiation that decays according to the $t^{-1.2}$ law. Each shield is machined to reduce the intensity by one-half. The plants are placed in concentric arcs around the source, and an entire series of exposures is given at one time for either FDS or constant-rate (CR) exposures.

Figure 1 shows the exposure-rate patterns for a total exposure of 5000 R for the treatments used in this study. The CR treatments simply extend for a specific time—in the present study this was for 1, 4, 8, or 16 hr. In the buildup and fallout-decay-simulation treatment (Bu + FDS), which is a close approximation to a true fallout situation, the exposure rate starts out at a low level, builds up in 51 min to a peak, and then decreases in a stepwise pattern over the exposure period. In the FDS treatment the exposure rate starts out very high and decreases in a similar stepwise fashion. The steps on the buildup and decay curves represent shields being raised or lowered, and, although this is a stepwise relation, the curve for accumulating exposure is fairly smooth, as shown in Fig. 2.

Experimental Procedure

In the first experiment seedlings of lettuce, *Lactuca sativa* 'Summer Bibb,' were exposed 26 days after sowing in 2-in. peat pots to the following treatments: (1) CR treatments for 1, 4, 8, or 16 hr or (2) changing-exposure-rate treatments given as either FDS or Bu + FDS for 36 hr. Fifteen exposures of 30 plants each, plus a nonirradiated control, were used for the 16-hr CR, FDS, and Bu + FDS treatments, and seven exposures of 10 plants each, plus a nonirradiated control, were used for the 1-, 4-, and 8-hr CR treatments. The experiment was carried out in early June 1969. The exposure rates for a total exposure of 5000 R were 5000, 1250, 625, and 312.5 R/hr for the 1-, 4-, 8-, and 16-hr treatments, respectively. The exposure rates for other total exposures

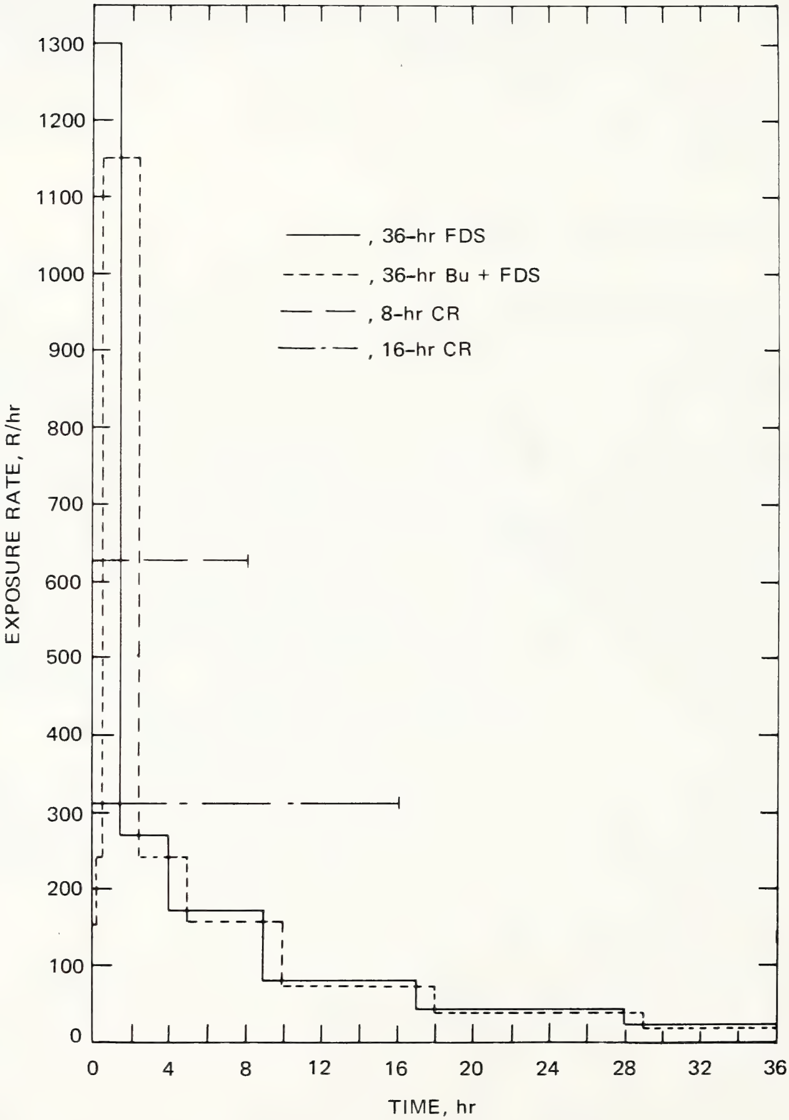

Fig. 1 Exposure-rate patterns for a total exposure of 5000 R for the treatments used.

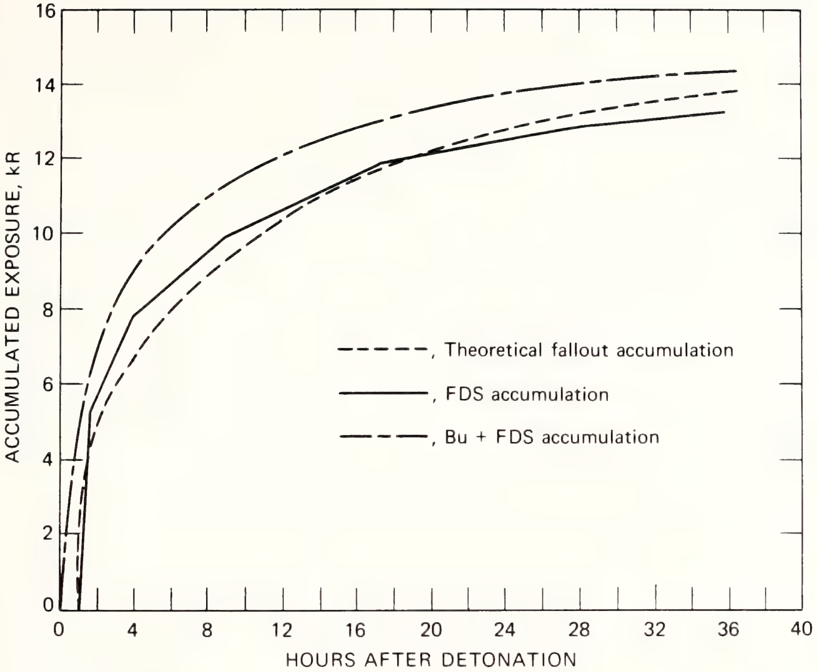

Fig. 2 **Accumulated exposures for the 36-hr FDS and 36-hr Bu + FDS at 1 m from the source compared with the theoretical accumulated exposures expected during the same period from decay according to the $t^{-1.2}$ law.**

varied in proportion to the exposure time. After irradiation the plants were transplanted to the field. Survival data were collected every other day until no more deaths attributable to the radiation occurred. Yield data measured as fresh weight of the aboveground portion of each plant were collected at the conclusion of the experiment.

In December 1969 seedlings of barley, *Hordeum vulgare* 'Mari,' 8 days after sowing in 2-in. peat pots, were irradiated with the following treatments: (1) CR treatments for either 8 or 16 hr or (2) a changing-exposure-rate treatment given as a 36-hr FDS. For each treatment there were 14 exposures of 10 plants each, plus a nonirradiated control. After irradiation the plants were transplanted into 6-in. clay pots and moved to a heated greenhouse. Survival data were collected three times a week until no more deaths attributable to the radiation occurred. At the conclusion of the experiment, the seed was harvested and weighed.

In February 1970 a similar experiment using the same treatments as used for barley was carried out with hard red spring wheat, *Triticum aestivum* 'Indus.' There were nine exposures of 10 plants each, plus a nonirradiated control for each treatment. The plants were transplanted into 4-in. clay pots and placed in a light- and temperature-controlled growth room. The light was cool white fluorescent and supplemental incandescent (approximately 1600 ft-c) on an 18-hr day, and the temperature was 68 ± 2°F at night and 72 ± 2°F during the day. Again survival data were collected three times a week, and the seed was collected and weighed at the end of the experiment.

An experiment to study the effect of exposure rate was conducted with germinating seeds of barley, *Hordeum vulgare* 'Himalaya.' Dry seeds (approximately 12% water content) were planted on blotters according to the method of Myhill and Konzak.[4] Irradiation began 24 hr after planting and the seeds were given an exposure of 1600 R delivered at 32 exposure rates ranging from 60 to 4800 R/hr for periods ranging from 26.6 hr to 19.8 min. Forty seedlings per exposure-rate treatment were used. After irradiation the seedlings were returned to a growth chamber and grown at 80°F under continuous fluorescent light. A constant high humidity was maintained in the chamber by bubbling air through a water reservoir. The height of the first leaf was measured 8 days after irradiation.

RESULTS

The results of the lettuce experiment are given in Fig. 3. The survival data (Fig. 3a) are shown on a probit plot of survival as percent of control against exposure for the three treatments. The graph shows the computer-fitted lines and actual data points. No difference was found between the Bu + FDS and the FDS treatments. Both treatments were more effective in reducing survival than the 16-hr CR treatment. The LD_{50} values for the three treatments were 4.79 ± 0.10 kR for FDS, 4.97 ± 0.12 kR for Bu + FDS, and 7.01 ± 0.12 kR for the 16-hr CR.

The yield data (Fig. 3b) show very little difference between the three treatments at the low exposures. At the higher exposures there was no difference between the results of the Bu + FDS and FDS treatments, but both were clearly more effective in reducing yield than the 16-hr CR treatment. A considerable amount of growth stimulation was evident at the lower exposures for all three treatments. This was found to be caused by the increased production of axillary growth, which contributed to the augmented fresh weight of the plant.

The survival results for the lettuce CR treatments are compared in Table 1. As the exposure time increased, the exposure required to produce the three given end points also increased. The nature of this relation is shown for the LD_{50} values in Fig. 4, where LD_{50} is plotted against the log of exposure time. There is little change in LD_{50} for the 1- and 2-hr treatments. As the exposure time is increased, however, LD_{50} increases almost with the square of the exposure time.

The results from the barley experiment are shown in Figs. 5 and 6. Figure 5 shows the probit plot of survival against exposure for the FDS and 16-hr CR treatments. The data are somewhat variable because only 10 plants per exposure

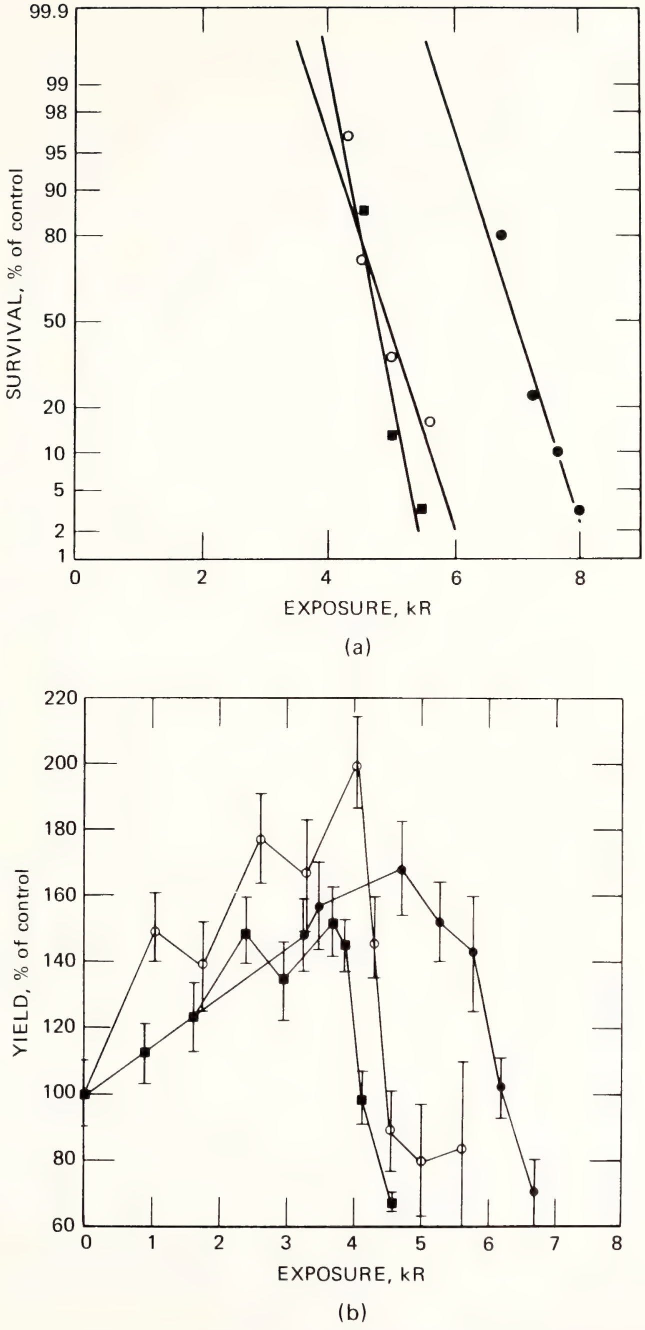

Fig. 3 (a) Probit plot of survival as percent of control vs. exposure for lettuce given 16-hr CR (●) and 36-hr FDS (■) and Bu + FDS (○) treatments. (b) Mean weight per treated plant as percent of control vs. exposure for lettuce for the same three treatments. ⊥ indicates ± standard deviation (Ref. 2).

Table 1

COMPARISON OF THE SURVIVAL END POINTS FOR 1-, 4-, 8-, AND 16-HR CR TREATMENTS FOR LETTUCE

	1-hr CR, kR ± S.D.*	4-hr CR, kR ± S.D.	8-hr CR, kR ± S.D.	16-hr CR, kR ± S.D.
LD_{10}	2.35 ± 0.06	3.03 ± 0.15	4.59 ± 0.11	6.39 ± 0.11
LD_{50}	2.57 ± 0.06	3.47 ± 0.10	5.03 ± 0.07	7.01 ± 0.07
LD_{90}	2.78 ± 0.08	3.90 ± 0.12	5.46 ± 0.13	7.64 ± 0.10

*The abbreviation S.D. is standard deviation.

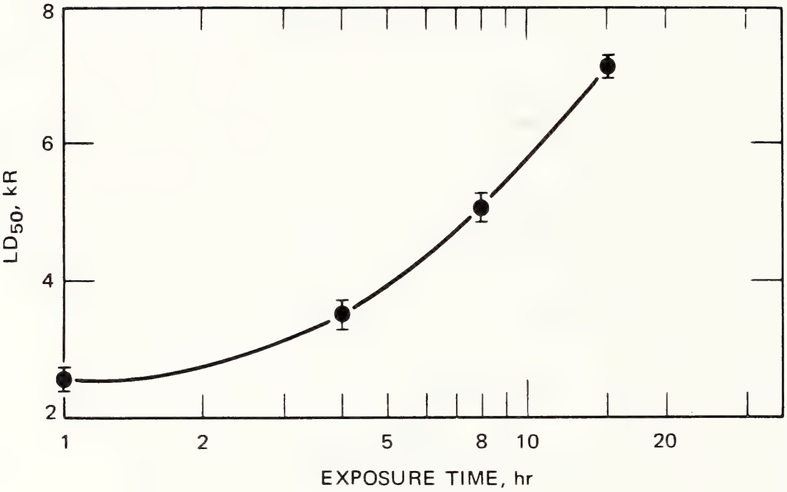

Fig. 4 **LD_{50} vs. log of exposure time for lettuce irradiated for 1, 4, 8, and 16 hr at constant rates. $\underset{\bot}{\overset{\top}{|}}$ indicates ± standard deviation.**

were used, but the results are consistent with those for the other species in showing the FDS treatment to be more effective in reducing survival than the 16-hr CR treatment. The yield data (Fig. 6) resemble the lettuce data (Fig. 3b) in that there is little difference between the FDS and 16-hr CR treatments at the lower exposures, but at exposures of 4 kR or more the CR treatment is clearly less effective in reducing yield. The 16-hr CR values are consistently above the FDS values although they are not always significantly different from them. Representative plants from the surviving exposures of the three treatments are shown in Fig. 7.

The probit plot of survival for wheat against exposure is given in Fig. 8, and again the FDS treatment was more effective in reducing survival than the 16-hr CR treatment. The yield data (Fig. 9) are similar to those for lettuce and barley

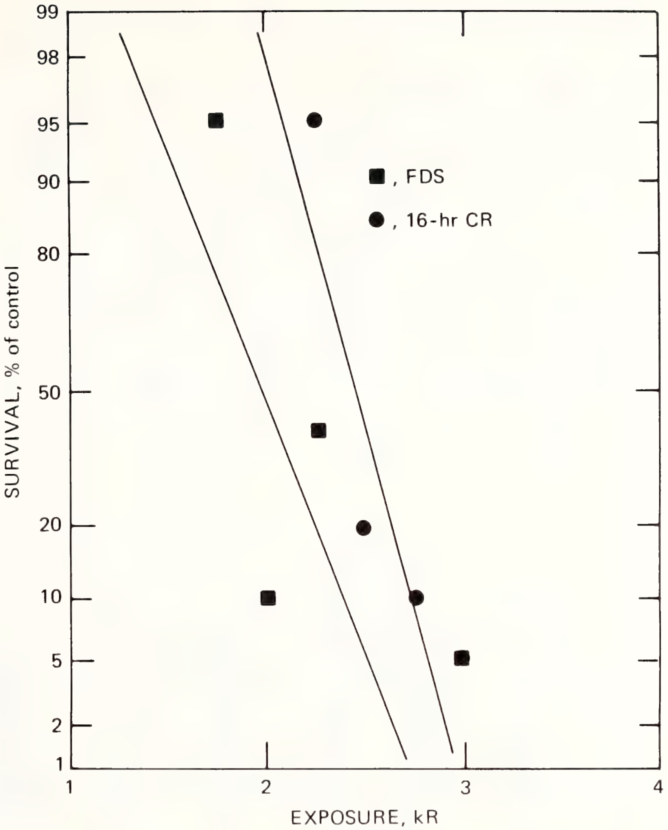

Fig. 5 Probit plot of survival as percent of control vs. exposure for barley given 36-hr FDS and 16-hr CR treatments.

in that the FDS treatment is more effective in reducing yield than the 16-hr CR treatment at the high exposures only. Representative plants of the surviving exposures from all treatments are shown in Fig. 10.

It became clear that a close relation might exist between the effects produced by 8-hr CR treatments and 36-hr FDS treatments. Therefore a comparison between these two treatments for both survival and yield was made for all three crops. This comparison is given for survival in Table 2 and Fig. 11 and for yield in Figs. 12 to 14. The effects of these two treatments are essentially the same, especially at the LD_{50}. Table 2 shows that the LD_{50} values for each crop were not significantly different at the 5% level. The situation is comparable when yield is the criterion of effect studied (Figs. 12 to 14).

The results of the barley exposure-rate experiment are given in Fig. 15. The injury increased in proportion to the log of exposure rate between 60 and

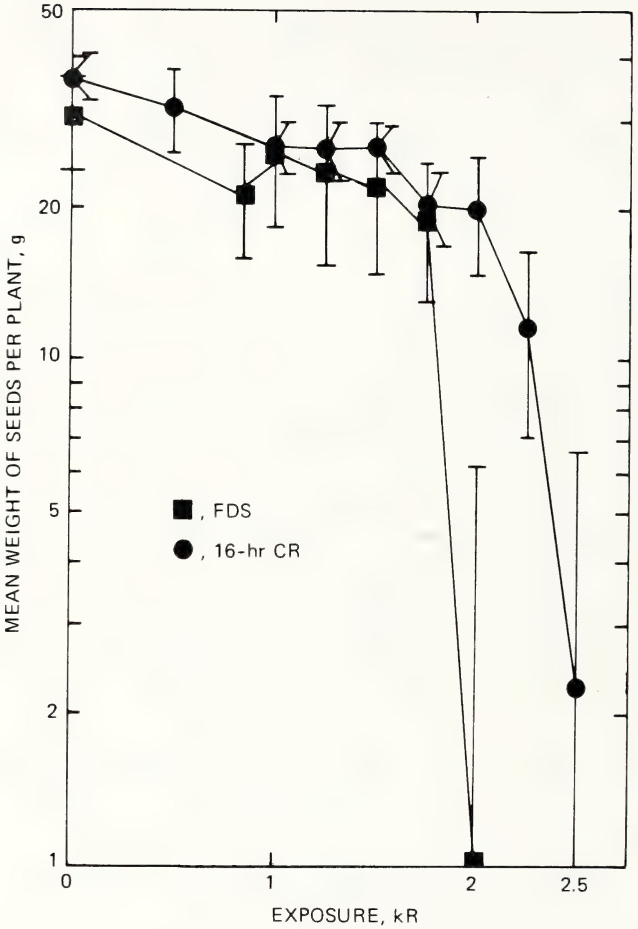

Fig. 6 Log mean weight of seeds per treated plant (in grams) vs. exposure for barley given 36-hr FDS and 16-hr CR treatments. ⊥ indicates 99% confidence interval.

1000 R/hr. However, very little change in the level of injury was found between 1000 and 4800 R/hr.

DISCUSSION

Most of the results given here may be explained on the basis of exposure rate; i.e., for the same total exposure, more damage occurs with high exposure rates than with low exposure rates. This, of course, is not a new concept in radiobiology, and the literature on the subject is too extensive to be reviewed in

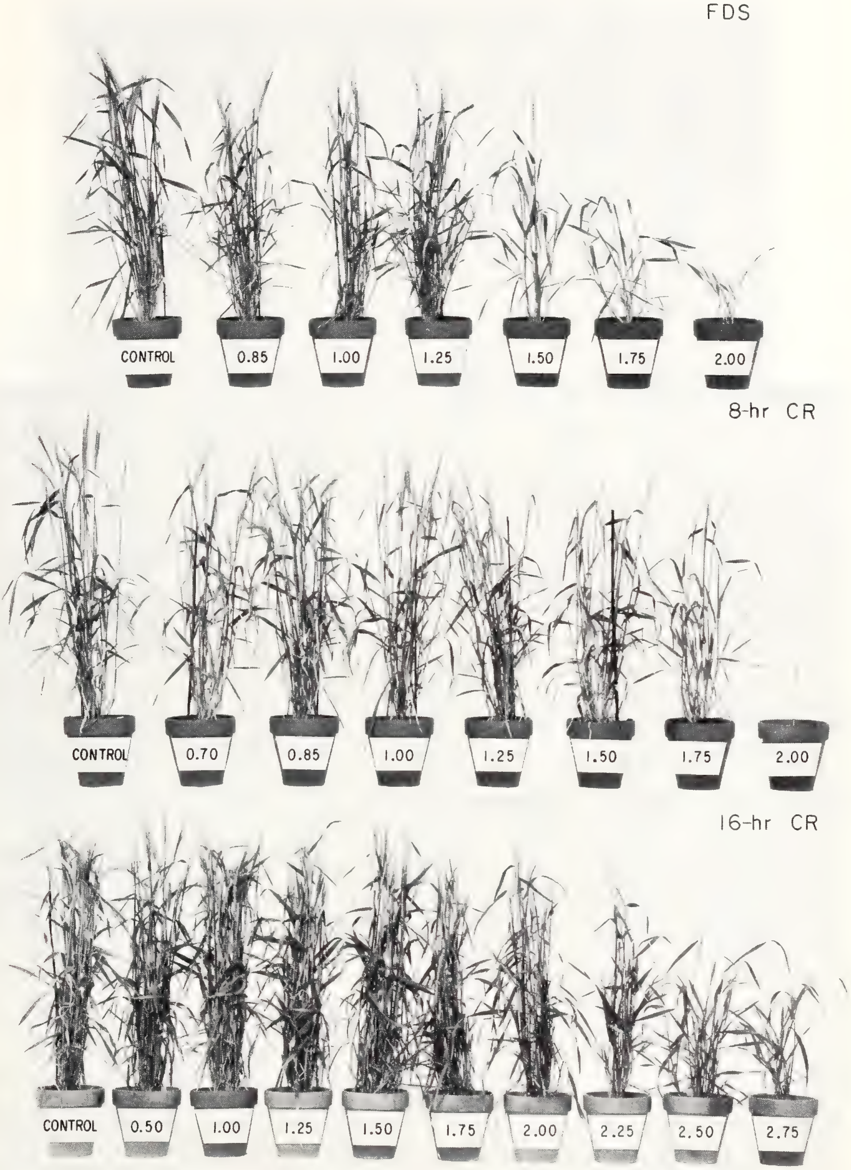

Fig. 7 Representative barley plants from the surviving exposures for 36-hr FDS, 8-hr CR, and 16-hr CR treatments. Exposures are given in kiloroentgens.

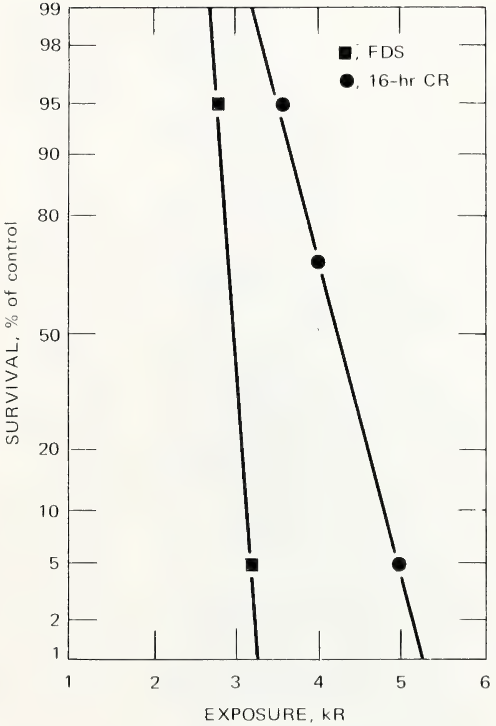

Fig. 8 Probit plot of survival as percent of control vs. exposure for wheat given 36-hr FDS and 16-hr CR treatments.

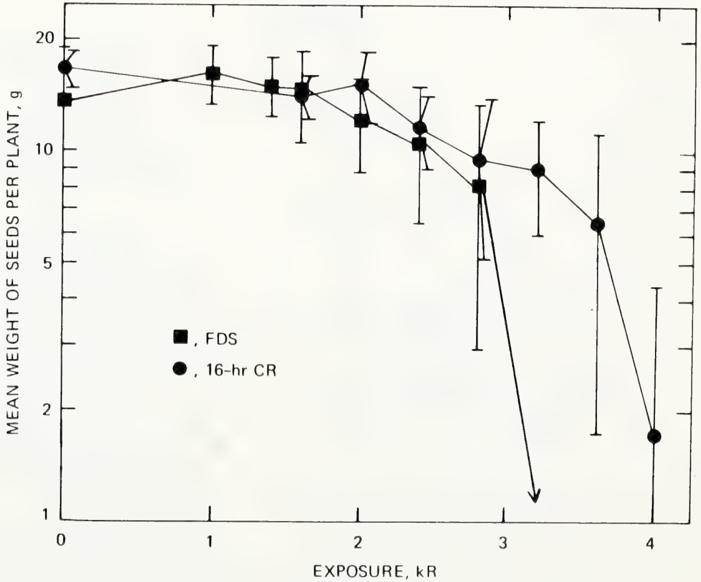

Fig. 9 Log mean weight of seeds per treated plant (in grams) vs. exposure for wheat given 36-hr FDS and 16-hr CR treatments. $\underset{\perp}{\top}$ indicates 99% confidence interval.

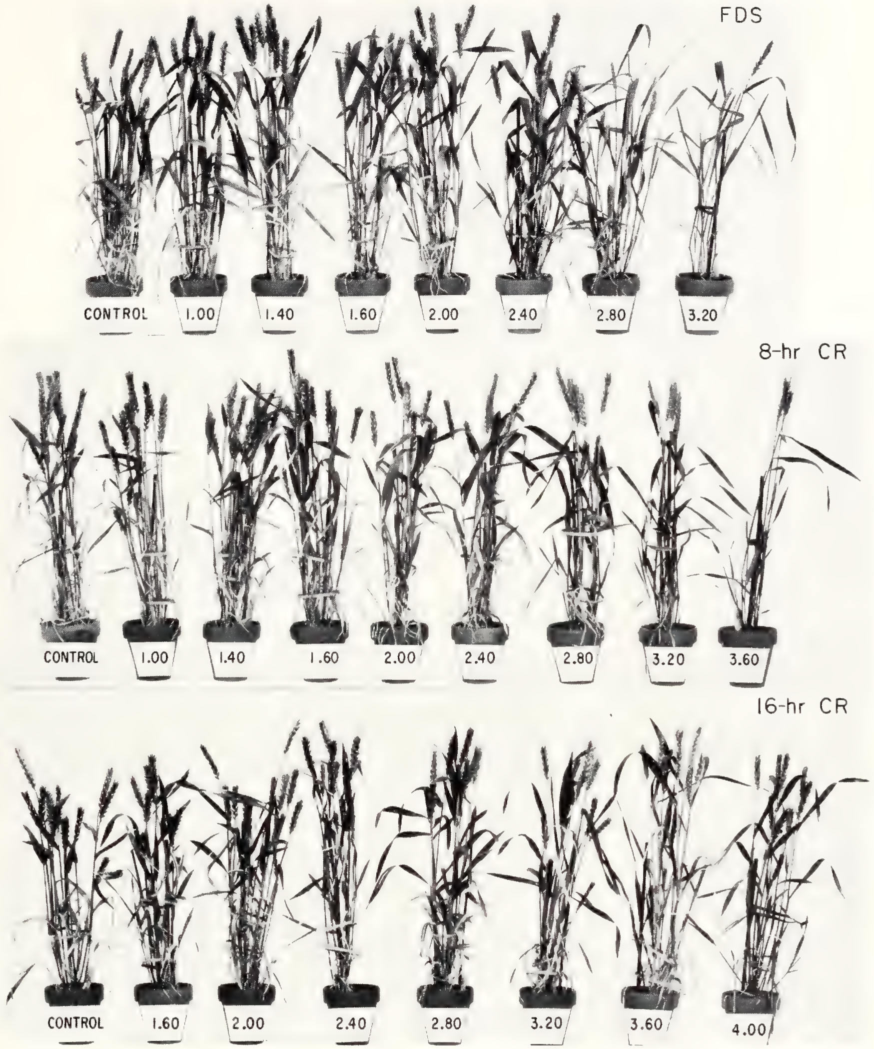

Fig. 10 Representative wheat plants from the surviving exposures of 36-hr FDS, 8-hr CR, and 16-hr CR treatments. Exposures are given in kiloroentgens.

depth here. In the majority of the published work, the effect measured increases with increasing exposure rate. This has been found for survival in *Solanum,*[5] barley,[6] *Neurospora,*[7] and aerobic HeLa cells;[8] for growth inhibition in *Vicia*[9,10] and barley roots;[11] for chromosome aberrations in pea[12] and barley seeds;[13] and for mutations in barley[6] and *Neurospora.*[7] An oxygen requirement has been shown for the expression of this exposure-rate effect.[8,14] This need is presumably due to the presence of repair mechanisms that require oxygen and

Table 2

COMPARISON OF LD_{50} VALUES FOR THE 8-HR CR AND FDS TREATMENTS FOR LETTUCE, BARLEY, AND WHEAT

Crop	Treatment	LD_{50}, kR ± S.D.*	
Lettuce	FDS	4.79 ± 0.05	N.S.†
	8-hr CR	5.03 ± 0.07	
Barley	FDS	1.99 ± 0.08	N.S.
	8-hr CR	1.91 ± 0.04	
Wheat	FDS	3.09 ± 0.71	N.S.
	8-hr CR	3.45 ± 1.12	

*The abbreviation S.D. is standard deviation.

†The abbreviation N.S. means not significant at the 5% level.

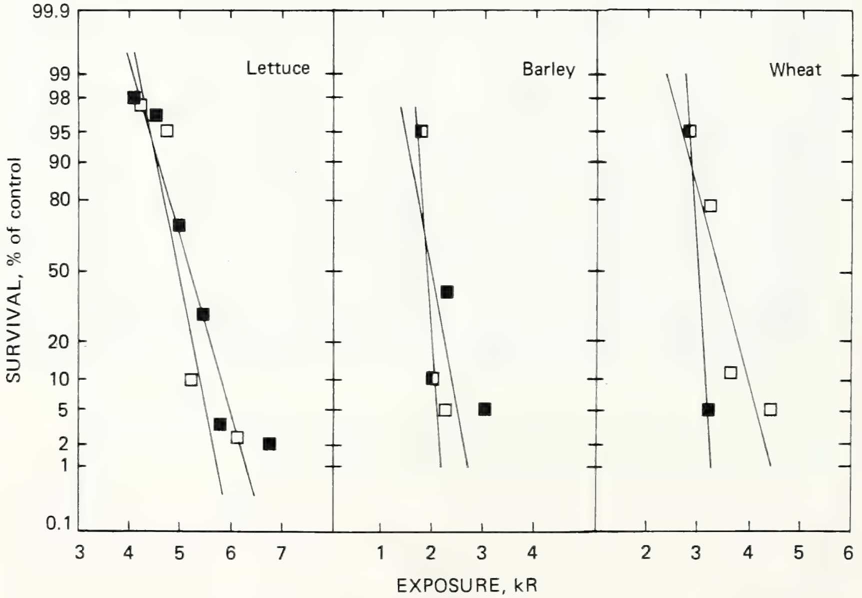

Fig. 11 Comparison of probit plots of survival as percent of control vs. exposure for lettuce, barley, and wheat given 8-hr CR (□) and 36-hr FDS treatments (■).

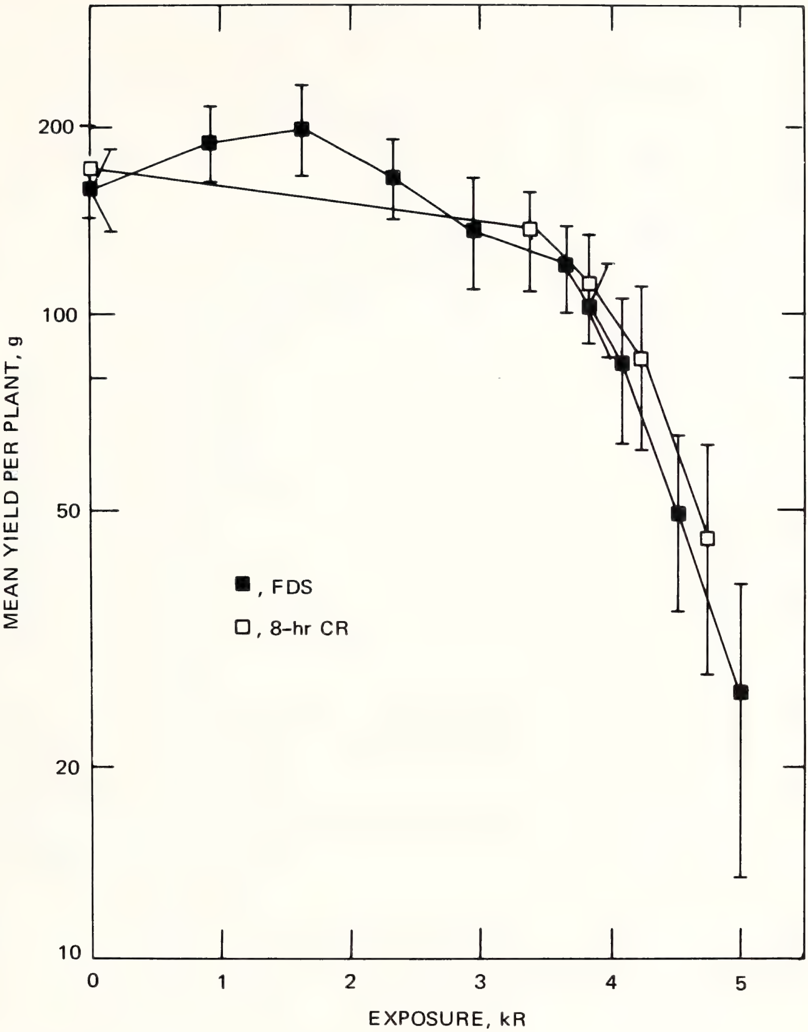

Fig. 12 **Log mean weight per treated plant (in grams) vs. exposure for lettuce given 8-hr CR and 36-hr FDS treatments. $\overset{\top}{\underset{\perp}{}}$ indicates 99% confidence interval.**

function most efficiently at low exposure rates. There are some limits to the exposure-rate effect, however. At very high exposure rates, further increases in rate do not bring about further increases in effect. This is in part a limitation of the system, as shown in the work of McCrory and Grun[5] where the 100% lethality level imposes an upper limit to the rate effect. This is not to say that an additional exposure-rate effect could not be shown, however; if the total exposure was decreased, there probably would be an additional exposure-rate

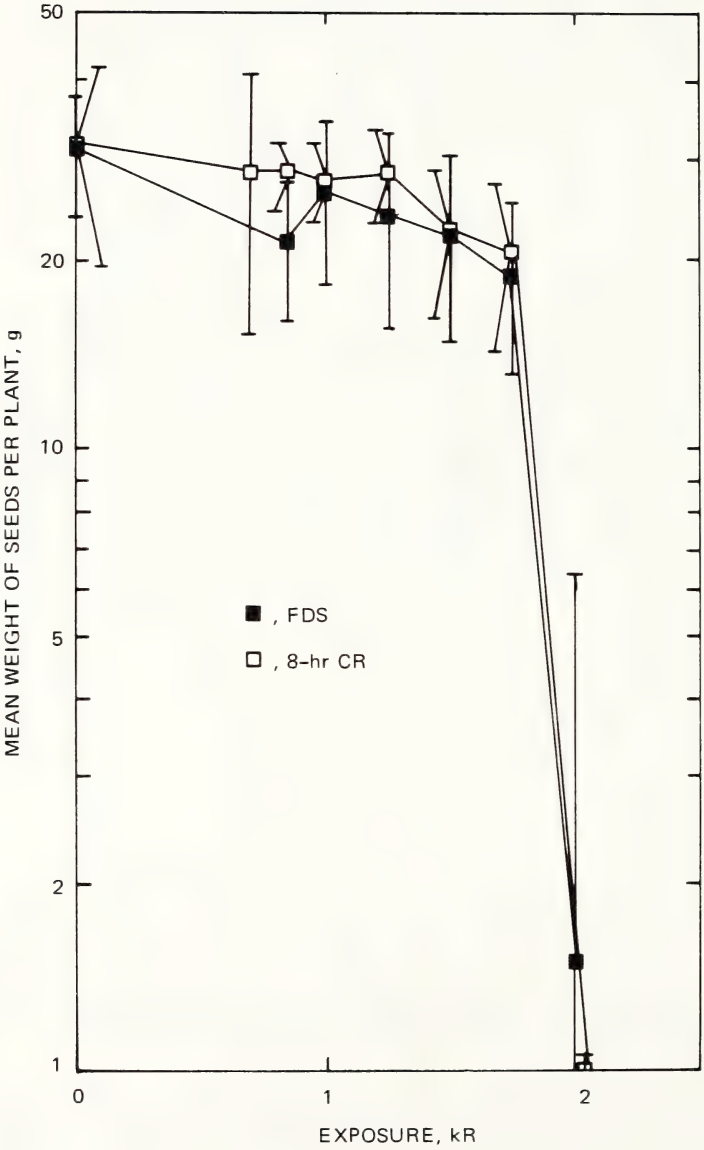

Fig. 13 Log mean weight of seeds per treated plant (in grams) vs. exposure for barley given 8-hr CR and 36-hr FDS treatments. ⊤⊥ indicates 99% confidence interval.

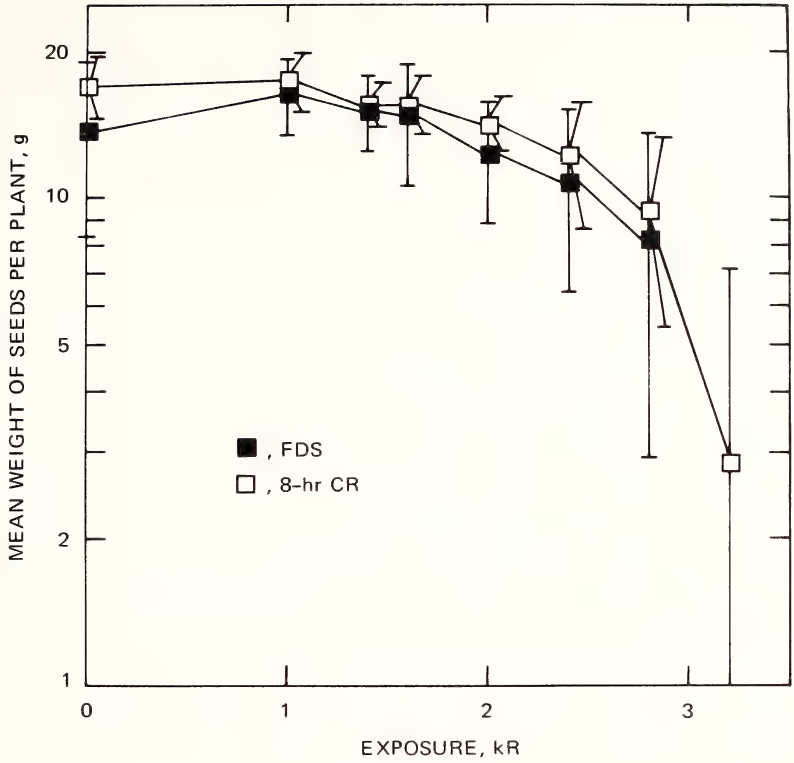

Fig. 14 Log mean weight of seeds per treated plant (in grams) vs. exposure for wheat given 8-hr CR and 36-hr FDS treatments. ⊤⊥ indicates 99% confidence interval.

effect. At the other end of the response curve, where the exposure rate is very low, a point is reached where no difference between irradiated and nonirradiated plants can be detected. This was observed by Hall and Bedford[10] for growth inhibition in *Vicia* roots and in some studies with chronic irradiation using many species.[15-17] This has led to the conclusion that, although the cumulative exposure is important, the rate at which that exposure is delivered is a more important factor.[17]

Thus there is substantial evidence in the literature for the exposure-rate effect reported here. We have observed an increasing effect with increasing exposure rate and have also reached the point in rate where no additional changes in effect occur with increasing rate. Experiments examining the response to lower exposure rates are under way. The most important factors controlling the specific exposure-rate effect are the species and criterion of effect used, the total exposure, and the environmental conditions during and after irradiation. Manipulation of these factors, e.g., lowering the total exposure, may allow one

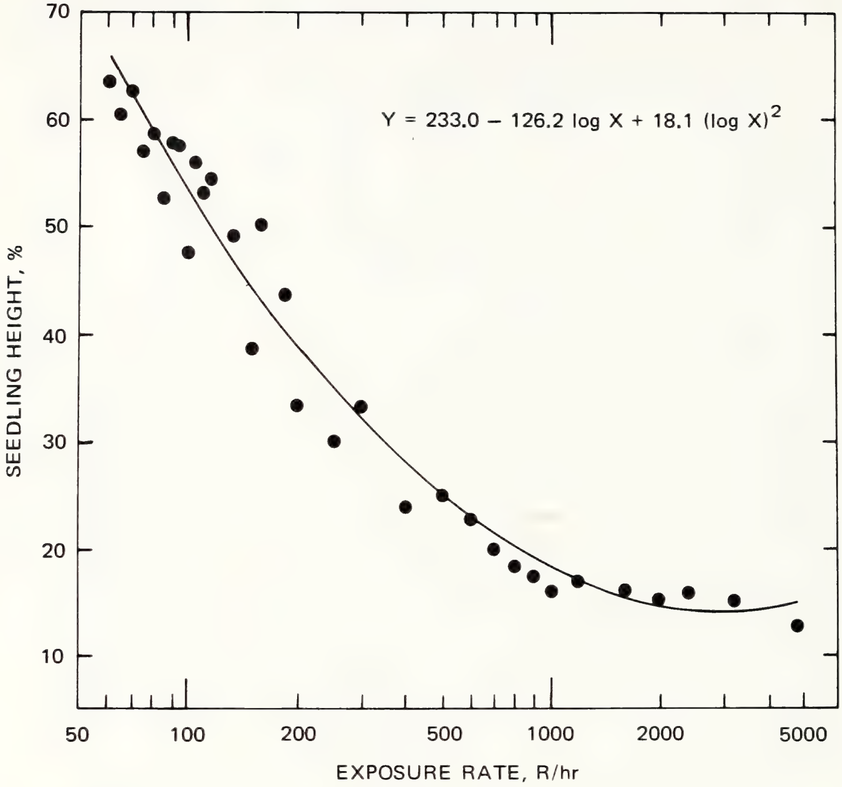

Fig. 15 Seedling height as percent of control vs. log of exposure rate for barley seedlings given a total exposure of 1600 R.

to demonstrate an effect at higher exposure rates since the capacity of the system to respond would be greater under conditions more conducive to expression of the effect.

We have reported both here and previously[1,2] that the FDS treatment is more effective in reducing survival and yield than the 16-hr CR treatment. The ratios of exposures at the LD_{50} for 16-hr CR to FDS are 1.43 for lettuce, 1.23 for barley, and 1.37 for wheat; these ratios agree well with the average of 1.4 for seven other species previously reported. The constant difference between the two treatments which was observed for survival was not observed for yield. At exposures up to the region of the FDS LD_{50}, little difference between the two treatments was observed. Above this exposure the yield for the FDS treatment falls off much more rapidly than the yield for the 16-hr CR treatment, and there is clearly a difference between the two. This difference in effectiveness is due to the very high exposure rates encountered in the early part of the FDS treatment. The average exposure rate in roentgens per hour (weighted for the shield timings) for a 5000-R exposure was calculated to be 791 R/hr for an FDS treatment as compared with 312.5 R/hr for the same total exposure from a 16-hr CR treatment. Thus the greater effectiveness of the FDS treatment can be explained by this difference of about 2.5 times in exposure rate. The fact that the survival and yield criteria for the FDS and Bu + FDS treatments are not greatly different is due to the use of essentially the same exposure-rate patterns for the two types of treatments (see Fig. 1).

The barley-seedling-height experiment shows that radiation damage increases with increasing exposure rate at rates below 1000 R/hr and provides additional support for our conclusion that the greater effectiveness of the FDS treatment is due to the initial high exposure rates. About 40% of the total exposure of 5000 R, which was lethal for lettuce, wheat, and barley, was given at 1300 R/hr. Although the criterion of effect studied was seedling-height reduction, it can be assumed that the survival and grain yield would also respond in a similar manner to variations in exposure rate. Thus the high overall exposure rate would be more than adequate to explain the increased effectiveness of the FDS treatment.

The similarity in effect between the 8-hr CR treatment and the FDS treatment is interesting from a practical standpoint. The exposure rates for the two treatments, compared for a 5000-R exposure, were found to be 625 R/hr for the 8-hr CR exposure and 791 R/hr for the FDS exposure. On this basis we would predict a similar level of effect for the two treatments if exposure rate played an important role in determining the level of damage. This finding is important since it implies that laboratories lacking the facilities to simulate fallout decay may obtain similar results by using 8-hr CR treatments. Although the 8-hr CR wheat data deviate somewhat from the FDS data for survival, the similarity between the FDS and the 8-hr CR data for all crops is very good, and relevant data on survival and yield for other crops can be made by using 8-hr CR treatments.

ACKNOWLEDGMENTS

We wish to thank Brenda Floyd, Susan S. Schwemmer, E. E. Klug, Leanne Puglielli, J. Newby, R. Sautkulis, Pamela Silimperi, and J. Bryant for assistance with the irradiations and data collection; R. A. Nilan for supplying the barley seed; and C. F. Konzak for supplying the wheat seed. The assistance of K. H. Thompson with statistics and Virginia Pond and Susan S. Schwemmer with critical comments on the manuscript is also acknowledged.

This research was carried out at Brookhaven National Laboratory under the auspices of the U. S. Atomic Energy Commission and the Office of Civil Defense, Department of the Army, Washington, D. C., under Project Order No. DAHC20-69-C 0167, Work Unit 3133E. Contracting office technical representative was D. W. Bensen. This report has been reviewed and approved for publication by the Office of Civil Defense. Approval does not signify that the contents necessarily reflect the views and policies of the Office of Civil Defense.

REFERENCES

1. A. H. Sparrow and Leanne Puglielli, Effects of Simulated Radioactive Fallout Decay on Growth and Yield of Cabbage, Maize, Peas and Radish, *Radiat. Bot.,* **9**: 77–92 (1969).
2. A. H. Sparrow, Brenda Floyd, and P. J. Bottino, Effects of Simulated Radioactive Fallout Buildup and Decay on Survival and Yield of Lettuce, Maize, Radish, Squash, and Tomato, *Radiat. Bot.,* **10**: 445–455 (1970).
3. G. M. Clark, F. Cheng, R. M. Roy, W. P. Sweaney, W. R. Bunting, and D. G. Baker, Effects of Thermal Stress and Simulated Fallout on Conifer Seeds, *Radiat. Bot.,* **7**: 167–175 (1967).
4. R. R. Myhill and C. F. Konzak, A New Technique for Culturing and Measuring Barley Seeds, *Crop Sci.,* **7**: 275–276 (1967).
5. J. McCrory and P. Grun, Relationship Between Radiation Dose Rate and Lethality of Diploid Clones of *Solanum, Radiat. Bot.,* **9**: 27–32 (1969).
6. A. Silvy and P. Pereau-Leroy, Effets genetiques d'irradiations gamma de courte durée et à différents débits de dose sur des plants d'orge (var. Piroline) en cours de développement, in *Induced Mutations in Plants,* Symposium Proceedings, Pullman, Wash., 1969, pp. 181–193, International Atomic Energy Agency, Vienna, 1969 (STI/PUB/231).
7. F. J. de Serres, H. V. Malling, and B. B. Webber, Dose-Rate Effects on Inactivation and Mutation Induction in *Neurospora crassa,* in Recovery and Repair Mechanisms in Radiobiology, Brookhaven Symposia in Biology Number 20, June 5–7, 1967, Upton, N. Y., USAEC Report BNL-50058, pp. 56–76, Brookhaven National Laboratory.
8. E. J. Hall, J. S. Bedford, and R. Oliver, Extreme Hypoxia; Its Effect on the Survival of Mammalian Cells Irradiated at High and Low Dose Rates, *Brit. J. Radiol.,* **39**: 302–307 (1966).
9. E. J. Hall and J. Cavanagh, The Oxygen Effect for Acute and Protracted Radiation Exposures Measured with Seedlings of *Vicia faba, Brit. J. Radiol.,* **40**: 128–133 (1967).
10. E. J. Hall and J. S. Bedford, A Comparison of the Effect of Acute and Protracted Gamma Radiation on the Growth of Seedlings of *Vicia faba.* Part 1. Experimental Observations, *Int. J. Radiat. Biol.,* **8**: 467–474 (1964).
11. L. Cercek, M. Ebert, and D. Greene, RBE, OER, and Dose-Rate Effects with 14 MeV Neutrons Relative to 300 kVp X-Rays in Barley Roots, *Int. J. Radiat. Biol.,* **14**: 453–462 (1969).
12. A. M. Akopyan, Effect of Different Types of Ionizing Radiations in Peas. II. Dose and Dose Rate Effects on the Frequency of Chromosomal Aberrations of Pea Seeds After Gamma Irradiation, *Genetika,* **7**: 34–38 (1967).
13. K. A. Filev, Postirradiation Seed-Storage Effect as Related to Irradiation Dose and Rate, *Dokl. Akad. Nauk SSSR,* **169**: 680–682 (1966).
14. D. L. Dewey, An Oxygen Dependent X-Ray Dose-Rate Effect in *Serratia marcescens, Radiat. Res.,* **38**: 467–474 (1969).
15. J. M. Bostrack and A. H. Sparrow, The Radiosensitivity of Gymnosperms. II. On the Nature of Radiation Injury and Cause of Death of *Pinus rigida* and *P. strobus* After Chronic Gamma Irradiation, *Radiat. Bot.,* **10**: 131–143 (1970).
16. G. M. Woodwell and A. H. Sparrow, Predicted and Observed Effects of Chronic Gamma Radiation on a Near-Climax Forest Ecosystem, *Radiat. Bot.,* **3**: 231–237 (1963).
17. J. E. Gunckel, A. H. Sparrow, Ilene B. Morrow, and Eric Christensen, Vegetative and Floral Morphology of Irradiated and Non-Irradiated Plants of *Tradescantia paludosa, Amer. J. Bot.,* **40**: 317-332 (1953).

FIELD STUDIES OF FALLOUT RETENTION BY PLANTS

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ABSTRACT

Several field studies on the retention by plants of local fallout particles (particles exceeding 44 μ in diameter) are summarized.

Although initial fractions of fallout intercepted varied as a function of plant-foliage characteristics and particle size, average initial retention values are similar for studies done with a wide variety of plants in different geographical regions.

Rapid losses of particles from foliage and other plant parts due to weathering occurred generally during the first week following initial particle deposition. Losses from tree species during this period were several times greater than losses from crop plants. In a period of 1 to 2 weeks following deposition all plants lost 90% or more of the fallout particles initially intercepted.

After about 3 weeks the loss of particles was relatively constant and proceeded at a slow rate (average weathering half-life of 21.3 ± 3.9 days) regardless of subsequent rain and wind conditions.

The formulation of realistic predictions of the biological effects of fallout on vegetation requires information on both the radiosensitivity of plants exposed to radiation in fallout geometries and the capacity of vegetation to intercept and retain fallout particles. Since about 64% of the total radiation dose from fallout is delivered during the first week after detonation of a nuclear device, initial interception, sites of deposition, and early losses of particles are critical events in estimating dose to contaminated plants.

This paper reviews some field studies on contamination of plants by local fallout and discusses the significance of these studies in the evaluation of short-term biological hazards involved in using nuclear devices for peaceful or military purposes.

Studies on retention of local fallout particles by plants have been made under both varying geographic and varying particle-source conditions. However, in many of these studies, the early losses of fallout from plants due to weathering have not been determined. This is particularly true of local fallout particles exceeding 44 μ in diameter. Deposits of particles exceeding 44 μ usually contain an appreciable fraction of fallout radioactivity, and they can represent a major source of radiation dose to plant tissues, although they may be briefly retained. Small particles constitute the bulk of radioactive debris deposited as worldwide fallout, but they lose much of their radioactivity via physical decay before deposition and are of greater biological significance as a major source of entry of radioactivity into food chains.

INITIAL RETENTION OF FALLOUT BY PLANTS

The initial retention of fallout by a given plant species depends on a number of factors. Such plant characteristics as surface area (mainly foliage), density, and surface characteristics of leaves are important variables. Meteorological conditions during deposition, particularly wind velocity and relative humidity, also influence initial retention. Finally, the size and amount of falling particles influence the degree to which plants are contaminated. Several field studies have been conducted in which the initial contamination of plants has been determined and related to one or more of these factors.

The initial retention of fallout by plants can be expressed in two ways. One is the foliage contamination factor (a_l) used by Miller:[1]

$$a_l = \mathrm{Ci}^0/m_i \qquad \text{sq ft/g} \tag{1}$$

where Ci^0 is the quantity in microcuries of radionuclide initially intercepted per gram of dry weight of foliage and m_i is the quantity in microcuries of radionuclide deposited per square foot of soil surface area. Another expression of initial retention is the fraction (F) of fallout which is intercepted by plants or foliage:

$$F = a_l w_l \tag{2}$$

where w_l is the biomass of foliage, or of the plant, in grams per square foot of soil surface area.

Values of a_l for plants sampled after nuclear tests have been smaller than values reported in other field tests where nonnuclear sources of fallout were applied. Miller,[1] reviewing a_l values for plants sampled following weapons tests, reported a range of 2×10^{-5} to 0.013 sq ft/g. Other estimates have been made by Martin.[2] Values from three weapons tests (Priscilla, Buffalo Round 2, and Sedan) ranged from 0.002 to 0.012, with an average of 0.004 sq ft/g. Most of the plant samples taken after nuclear detonations were collected several days after initial deposition of fallout or after some losses due to weathering had occurred. Also, results from test-site fallout fields were usually obtained in areas of light to moderate fallout.

Plant contamination values derived from several field studies with local fallout and, where samples were taken before appreciable weathering, are fairly consistent. The a_l values for a variety of different plant species were taken by Miller[1] in Costa Rica following deposition of fallout from the Irazu volcano. A median value of 0.05 sq ft/g was reported for dry exposure conditions and particles having a median diameter between 50 and 100 μ. In studies at Oak Ridge National Laboratory, Witherspoon and Taylor[3] reported an average a_l value of 0.057 ± 0.024 sq ft/g for five species of crop plants treated with 88- to 175-μ diameter particles. In similar studies[4] values of 0.035 and 0.005 sq ft/g were reported for oak and pine tree foliage, respectively. Values for relatively small-leaved plants such as pine[4] (0.005), lespedeza[3] (0.010), and fescue grass[5] (0.011) tend to be smaller than those for large-leaved plants.

Therefore an a_l value of 0.05 sq ft/g seems reasonable for calculating initial beta-exposure doses to plants in areas of local, dry fallout deposition (or where particles exceed 50 μ in diameter). A value of about 0.01 may represent a good estimate for most narrow-leaved plants. Under damp conditions (relative humidity greater than 90%), or where foliage surfaces are wet, a_l values have been reported to increase by an average of two to four times those obtained under dry conditions.[1,6]

Reported values of F, initial fraction of fallout intercepted by plants relative to amount deposited per open soil surface area, are summarized in Table 1.

Table 1

INITIAL RETENTION OF SIMULATED FALLOUT DEPOSITED IN AN ACUTE MODE UNDER DRY CONDITIONS

Plant	Retention, %			Foliage area/Soil surface area, sq ft/sq ft	Plant density, g/sq ft of soil
	44- to 88-μ particles	88- to 175-μ particles	175- to 350-μ particles		
White pine[4]*		24.2			44.6
Red oak[4]		34.9			9.9
Squash[3]	100.0	88.5		1.72	6.4
Soybean[3]	100.0	100.0		3.11	11.4
Lespedeza[3]	7.5	1.9		0.51	1.9
Peanut[3]	9.8	5.8		0.91	4.4
Sorghum[3]	48.9	10.8		1.25	5.4
Fescue[5]	45.4	19.6			17.4
Pasture grass[6]		7.4	5.5		9.2
Alfalfa[6]		23.0	5.0		19.5
Corn[6]		44.0			

*Reference number.

Initial retention can be seen to vary both with plant-foliage types and with particle size. Where retention values for different particle sizes can be compared, there is an average of two to three times less initial retention when particle size range is increased by a factor of 2. This is particularly evident in plants having small leaves and small foliage surface area relative to soil surface area. Mass loading of particles used in the studies summarized in Table 1 varied from about 0.5 to 13.6 g of particles per square foot of open soil surface. In one series of studies[6] where mass loading was varied, initial retention was found to be independent of mass loading over this range.

Sites of retention other than foliage can be important in determining biological effects of fallout radiation on plant species. Table 2 gives the average fraction of total initial retention associated with various plant parts. For these particular species the foliage intercepted most of the fallout, but small fractions were intercepted by radiosensitive structures such as flowers and buds. In the white pine, a relatively radiosensitive plant species, a large fraction of fallout is trapped in clusters of buds on the ends of branches. Since these buds contain meristematic tissues, which are the most radiosensitive parts of the vegetating plant, these trapping sites represent critical regions. Particles trapped in these structures also are retained longer than particles intercepted by pine foliage.[4] Flowers also may intercept small fractions of fallout. In squash plants, Table 2, initial interception by flowers of particles 44 to 88 μ in diameter was less than that of 88- to 175-μ particles applied at the same mass loading. The larger particles may have had a tendency to bounce or roll off foliage into open flowers, whereas smaller particles were more efficiently intercepted by foliage and stem surfaces. Smaller particles, in the 44- to 88-μ range, were intercepted by vertical structures, such as sorghum stalks, with much greater efficiency than

Table 2

FRACTION OF TOTAL INITIAL RETENTION IN PLANT PARTS*

Plant	Plant part	Fraction of 44- to 88-μ particles	Fraction of 88- to 175-μ particles
Squash	Stem	0.051	0.037
	Flowers	0.007	0.032
	Foliage	0.942	0.931
Sorghum	Stalk	0.259	0.086
	Foliage	0.741	0.914
White pine	Bud clusters		0.160
	Foliage		0.840

*Fallout applied under dry conditions at a mass loading of from 4.5 to 6.6 g per square foot of open soil surface.

larger particles. Grasslike plants such as sorghum, corn, and fescue have effective particle-trapping sites in the leaf axils, angles between the leaves and stems.[3,5,6] Unless particles contain enough radioactivity to produce damage to tissue from contact doses, however, these trapping sites may not be biologically important since they are somewhat removed from more radiosensitive meristematic regions.

LOSS OF FALLOUT FROM PLANTS DUE TO WEATHERING

The major meteorological factors that influence retention of fallout by plants are wind speed and rainfall. Estimation of early losses of fallout particles from plants, particularly for the first week following initial deposition, is critical in determining dose to contaminated plants. Results from studies on retention indicate that concentrations of radionuclides on fallout-contaminated plants can be expected to decrease at rates significantly higher than would be predicted on the basis of physical, radioactive decay. Beta-radiation-exposure geometries may be expected to change rapidly from a contact to a bath mode of exposure.

Loss of fallout from foliage during the first day following deposition is rapid under dry conditions with relatively gentle wind speeds. Table 3 illustrates prompt losses for 10 plant species studied under similar conditions of deposition mode and weather. In most cases these losses amount to 50% or more of the amounts initially intercepted. Studies with smaller particles (44 to 88 μ) have indicated that first-day losses are as great as with 88- to 175-μ particles.[3] Rapid particle loss from other plant structures also may be expected. Table 4 gives

Table 3

PROMPT LOSSES OF 88- TO 175-μ PARTICLES FROM FOLIAGE

Plant	Time after deposition, hr	Initial interception remaining, %	Wind, mph	Rain, in.
Corn[6]*	24	94	0 to 20	
Alfalfa[6]	24	82	0 to 20	
Squash[3]	36	52	0 to 5	
Soybean[3]	36	49	0 to 5	
Sorghum[3]	36	90	0 to 5	
Peanut[3]	36	44	0 to 5	
Lespedeza[3]	36	74	0 to 5	
Fescue[5]	18	34	0 to 1.5	
White pine[4]	1	90	0 to 12	
White pine[4]	24	6.3	0 to 15	0.9
Red oak[4]	1	9.5	0 to 12	
Red oak[4]	24	0.4	0 to 15	0.9

*Reference number.

Table 4

LOSSES OF 88- TO 175-μ PARTICLES FROM SQUASH AND SORGHUM DUE TO WIND ACTION

Time, days	Wind, mph	Retention, %*				
		Squash			Sorghum	
		Foliage	Flowers	Stem	Foliage	Stalk
0.5	0 to 5	86.2	37.5	93.0	96.8	53.4
1.5	0 to 5	52.4	14.8	90.0	90.0	33.8
7	0 to 7	36.0	10.0	44.1	47.0	2.6

*Percent of initial interception value.

retention values for stem and flowers of squash and for sorghum stalks. Rate of loss of particles from squash foliage was greater than that from the stem over a period of 1 week after initial deposition. It is probable that stems, which are prostrate and under the large leaves, intercepted some of the particles dislodged from foliage by gentle winds during this period. The more rapid loss rate from flowers was due, in this case, to wilting and loss of petals during this period—a phenological event. Rapid losses from structures such as the vertical stalks of sorghum were expected.

Some generalizations concerning the probable retention of fallout by trees vs. agricultural plants may be made. Table 5 gives average foliage-retention values for five crop species[3] that vary in growth habit and leaf-surface characteristics and for two tree species that represent very common tree-foliage types. Initial

Table 5

AVERAGE RETENTION* OF 88- TO 175-μ PARTICLES BY PLANTS UP TO 5 WEEKS AFTER DEPOSITION

Time after application, days	Average retention of five crop species,[3] %	Accumulated rainfall, in.	Average retention, %		Accumulated rainfall, in.
			White pine[4]	Red oak[4]	
0.04		0	91.0 ± 10.0	9.50 ± 0.81	0
1	74.0 ± 8.3	0	6.3 ± 0.8	0.39 ± 0.06	0.90
1.5	61.8 ± 8.7	0	4.5 ± 0.4	0.25 ± 0.04	0.90
7	33.0 ± 4.6	0.25	2.5 ± 0.2	0.02 ± 0.003	1.30
14	9.4 ± 4.7	1.28	2.1 ± 0.2	0.015 ± 0.001	1.43
21	2.7 ± 1.2	2.67	1.9 ± 0.3	0.012 ± 0.001	1.47
28	2.6 ± 1.5	2.67	1.6 ± 0.2	0.010 ± 0.002	2.88
35	2.6 ± 1.6	2.67	1.2 ± 0.1	0.010 ± 0.002	3.46

*Average percent of initial interception ± 1 standard error.

particle losses, up to 1 week, were much greater for the trees. The data for trees reflect, however, the effects of one rain which fell 12 hr after initial deposition. The losses from crop plants for the first 6 days were due to wind action only. Nevertheless, with comparable rainfall for the duration of these studies, up to 5 weeks after deposition, losses from trees were greater. Smooth-leaved trees, such as the red oak, retained only a small fraction of the initial deposition after 1 week. All these plants lost the major portion (90%) of the fallout in 1 to 2 weeks, a period in which the major portion of fallout-radiation dose is delivered. Not only did the trees lose particles faster than the crop species tested but also, from the standpoint of dose, this loss is more important for trees. Particle loss can also be interpreted as a change in beta-exposure geometry from a contact to a bath mode, and the most radiosensitive structures (meristems and flowers) are located at greater distances from the ground in trees than in crop plants. Therefore bath doses from fallout on the ground would be less serious, impart less dose, to trees than to crop plants because of their relatively greater height.

Retention of particles beyond 2 weeks was relatively stable for trees and crop plants regardless of amount of wind and rainfall. By this time most of the fallout has probably become trapped in sites upon which subsequent weathering has little effect. Retention characteristics after this time may be important from the standpoint of chronic low-level dose or transfer into food chains.

Data on fallout retention of plants or plant parts plotted vs. time typically take the form of an exponential curve. In the calculation of half-lives, however, it is difficult to express these data in terms of a single weathering or effective half-life.[4] Rapid particle losses during the first day or week and subsequent loss-rate changes after early weathering imply that retention data should be compartmentalized into appropriate time components for half-life analyses. Table 6 gives weathering half-lives for 88- to 175-μ particles on seven species of plants. These half-lives are given for three time components: initial deposition to 1.5 days, when very rapid loss rates occur; 1.5 to 14 days; and 14 to 33 days, when loss rates tend to stabilize at a very slow rate. Averaging these weathering half-lives gives some indication of general particle retention for a wide variety of plants. Such averages may be useful in dose calculations for periods up to several weeks following deposition.

Environmental half-lives (e.g., half-life rates of loss due to causes other than radioactive decay) of radionuclides on fallout-contaminated plants were reported by Martin[7] for plants in the Sedan fallout field. Bartlett et al.[8] reported these values for plants sprayed with fission-product solutions. In the Sedan fallout field from 5 to 30 days after detonation, the environmental half-lives for fallout ^{89}Sr and ^{131}I were 28 and 13 to 17 days, respectively.[7] Fission products sprayed on grass exposed to wind and rain up to 60 days had an average environmental half-life of about 14 days.[8]

The average weathering half-life for the 14- to 33-day time component in Table 6 is 21.3 ± 3.9 days. Thus it appears that weathering or environmental half-life values for different kinds of vegetation growing in different geographical

Table 6

WEATHERING HALF-LIVES OF 88- TO 175-μ PARTICLES ON FOLIAGE FOR THREE TIME COMPONENTS

Plant	Half-life for 0 to 1.5 days, days,	Rain, in.	Half-life for 1.5 to 14 days, days,	Rain, in.	Half-life for 14 to 33 days, days,	Rain, in.
White pine[4]*	0.69	0.9	13.09	1.43	26.14	1.97
Red oak[4]	0.64	0.9	6.11	1.43	42.58	1.97
Squash[3]	1.62	0	7.36	1.28	15.06	1.39
Soybean[3]	1.47	0	7.19	1.28	15.97	1.39
Sorghum[3]	4.10	0	7.43	1.28	19.43	1.39
Peanut[3]	1.33	0	15.71	1.28	16.07	1.39
Lespedeza[3]	2.88	0	7.55	1.28	14.07	1.39
Average ±1 standard error	1.82 ± 0.48		9.20 ± 1.33		21.33 ± 3.96	

*Reference number.

regions may be similar after the rapid initial losses during the first week or so have occurred. The average values for different species (1.82 ± 0.48 days for the 0- to 1.5-day component and 9.20 ± 1.33 days for the 1.5- to 14-day component) and the ranges given in Table 6 suggest that weathering half-lives may differ only by a factor of slightly over 1 to about 6.5 between species during the periods of rapid initial particle loss.

The similarities in results from field studies in which particle size approximates that of local fallout are striking. Both initial contamination factors, such as the a_l value, and weathering half-lives for time components may be in close enough agreement so that the use of averages, such as those presented here, would give reasonable estimates of dose from fallout when used in appropriate models.

ACKNOWLEDGMENT

This research was sponsored by the U. S. Atomic Energy Commission under contract with Union Carbide Corporation and the Office of Civil Defense, Department of Defense.

REFERENCES

1. C. F. Miller and H. Lee, Operation Ceniza-Arena. Part One, SRI Project MU-4890, Stanford Research Institute, 1966.

2. W. E. Martin, Early Food-Chain Kinetics of Radionuclides Following Close-In Fallout from a Single Nuclear Detonation, in *Radioactive Fallout from Nuclear Weapons Tests,* Germantown, Md., Nov. 3–6, 1964, A. W. Klement (Editor), AEC Symposium Series, No. 5 (CONF-765), 1965.
3. J. P. Witherspoon and F. G. Taylor, Interception and Retention of a Simulated Fallout by Agricultural Plants, *Health Phys.,* **19**: 493-499 (1970).
4. J. P. Witherspoon and F. G. Taylor, Retention of a Fallout Simulant Containing ^{137}Cs by Pine and Oak Trees, *Health Phys.,* **17**: 825-829 (1969).
5. R. C. Dahlman, in Progress Report in Postattack Ecology. Interim Report, USAEC Report ORNL-TM-2983, Oak Ridge National Laboratory, December 1970.
6. J. E. Johnson and A. I. Lovaas, Deposition and Retention of Simulated Near-In Fallout by Food Crops and Livestock, Technical Progress Report 3223C, Colorado State University, 1969.
7. W. E. Martin, Losses of ^{90}Sr, ^{89}Sr and ^{131}I from Fallout-Contaminated Plants, *Health Phys.,* **4**: 275-284 (1964).
8. B. O. Bartlett, L. F. Middleton, G. M. Milbourn, and H. M. Squire, The Removal of Fission Products from Grass by Rain, in Surveys of Radioactivity in Human Diet and Experimental Studies, British Report ARCRL-5, pp. 52–54, 1961.

ECOLOGICAL EFFECTS OF ACUTE BETA IRRADIATION FROM SIMULATED-FALLOUT PARTICLES ON A NATURAL PLANT COMMUNITY

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ABSTRACT

Simulated-fallout particles overcoated with ^{90}Y were applied at two levels of activity to granite-outcrop plant communities. The experiment resembled conditions expected at a site 170 miles downwind of a 2.5-Mt detonation with a wind velocity of 15 mph. Mean community dose levels were 7000 and 4000 rads. In the 7000-rad communities, the ratio of mean ground-surface dose (8770 rads) to mean canopy dose (5092 rads) was 1.7. In the 4000-rad communities, the mean ground-surface dose (4824 rads) was 1.6 times higher than the mean canopy dose (2996 rads).

In the 7000-rad communities, the death of 46% of all terminal buds in the dominant *Viguiera porteri* resulted in a 37% height-growth reduction, a compensatory lateral branch development, a 16% reduction in community biomass, and a lower, more clumped, vertical distribution of leaves in the canopy. Comparison with earlier studies indicated that acute beta irradiation may be twice as effective as chronic gamma irradiation at equivalent total doses in causing height-growth reduction in *V. porteri.*

No radiation-induced change in the metabolism of the outcrop ecosystem was detected through measurements of CO_2 exchange 43 days after fallout dispersal. The mean rate of net production on clear days in both July and September (9:30 a.m. to 4:40 p.m.) was 1.2 g C/m^2/hr. Early nighttime rates of respiration (9:30 to 10:10 p.m.) averaged 2.9 g C/m^2/hr in July and 2.2 g C/m^2/hr in September.

Only since 1959 have the effects of ionizing radiation on entire plant communities and ecosystems been experimentally investigated. McCormick's study of a granite-outcrop plant community[1] and Woodwell's study of a Long Island forest[2] were among the first investigations of radiation effects in natural plant communities. A common finding of these and subsequent studies has been that ecosystems respond to radiation stress much as they do to other

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THE SIGNIFICANCE OF LONG-LIVED NUCLIDES AFTER A NUCLEAR WAR

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ABSTRACT

The radiation doses from the long-lived nuclides ^{90}Sr and ^{137}Cs, to which the surviving population might be exposed after a nuclear war, are considered using a new evaluation of the transfer of ^{90}Sr into food chains.

As an example, it is estimated that, in an area where the initial deposit of near-in fallout delivered 100 R/hr at 1 hr and there was subsequent worldwide fallout from 5000 Mt of fission, the dose commitment would be about 2 rads to the bone marrow of the population and 1 rad to the whole body. Worldwide fallout would be responsible for the major part of these doses.

In view of the possible magnitude of the doses from long-lived nuclides, the small degree of protection that could be provided against them, and the considerable strain any such attempt would impose on the resources of the community, it seems unrealistic to consider remedial measures against doses of this magnitude. Civil-defense measures should be directed at mitigating the considerably higher doses that short-lived nuclides would cause in the early period.

It is now widely recognized that long-lived fission products would make a negligible contribution to the radiation exposure of the population in heavily contaminated areas shortly after a nuclear attack. The external radiation dose would usually be dominant, and, if simple precautions were taken to avoid the superficial contamination of foodstuffs, the entry of ^{131}I into milk would cause the only important problem of dietary contamination. Thus, for example, infants probably would not receive doses of more than 0.1 rad to bone marrow from ^{90}Sr nor more than 0.01 rad from ^{137}Cs in the weeks after a nuclear attack if they were fed continuously with milk produced in an area where the external dose rate at 1 hr after detonation had been 100 R/hr. Doses to the thyroid from ^{131}I might, however, exceed 200 rads.[1] Considerably higher doses from dietary contamination were expected until it became evident that the physical properties of near-in fallout much reduce the entry of radioactivity into food chains.

In more lightly contaminated areas, especially where deposition does not occur for many hours or days, internal radiation would give rise to a larger fraction of the total radiation dose, partly because short-lived nuclides would have decayed before fallout descended and partly because fission products contained in the more finely divided and soluble distant fallout enter food chains more readily. The relative contributions of ^{131}I, ^{90}Sr, and ^{137}Cs to the internal radiation dose would, however, be comparable to those in near-in localities.

Civil defense planning is naturally concerned primarily with this early period when external radiation is dominant, but this is not the whole story. Years after a nuclear war, long-lived nuclides will remain in the soil and will continue to descend in worldwide fallout. Therefore two questions are relevant: (1) What radiation doses will be received from these sources by the survivors of a nuclear war? (2) Is it prudent and realistic to prepare plans for long-term remedial action against the contamination of agricultural produce? This paper discusses these questions in relation to dietary contamination.

For obvious reasons the long-term problems will be caused largely by ^{90}Sr, the extent to which it will enter food chains from the soil many years after deposition being a question of major relevance. It is therefore appropriate to review information on this question in some detail.

ENTRY OF ^{90}Sr INTO FOOD CHAINS FROM THE SOIL

Our understanding of the behavior of ^{90}Sr in the soil has been much aided by experiments in which weapon debris or measured quantities of ^{85}Sr, ^{89}Sr, or ^{90}Sr have been incorporated into the soil, but quantitative relations that can be confidently applied to wide areas cannot be obtained from these small-scale studies. The best approach is to analyze the results of surveys of deposition of worldwide fallout and contamination of foodstuffs, thus partitioning the contamination of food between direct contamination (i.e., the retention of the recent deposits on vegetation) and that resulting from uptake from the soil. Many of the uncertainties that arise in extrapolating from limited data are thus avoided.

The first analysis of this type was made at the initiative of Tajima by the United Nations Scientific Committee on the Effects of Atomic Radiation[2] (UNSCEAR) in 1958. Like most subsequent studies, his work was concerned with the contamination of milk, because of its importance in the transfer of ^{90}Sr to human diet. Using the available survey data on the contamination of milk and the deposition of fallout, he attempted to solve simple empirical equations of the following type:

$$C = p_r F_r + p_d F_d \qquad (1)$$

where C = annual mean ratio of ^{90}Sr to calcium in milk (pCi ^{90}Sr/g Ca)
F_r = deposit of ^{90}Sr in the year in question (mCi/km^2)
F_d = cumulative total deposit after allowance for radioactive decay (mCi/km^2)
p_r, p_d = proportionality factors

Our discussion of the use of this and other procedures is concerned mainly with relations in the United Kingdom, but, as will be shown later, the situation there seems relatively typical of temperate regions. From survey data up to 1961, p_r and p_d were estimated to be 0.76 and 0.19 (Ref. 3). Annual milk levels calculated on this basis for past years agreed reasonably with those observed (Fig. 1b), but the defects of Eq. 1 were nonetheless obvious. First, the equation assumed that all ^{90}Sr entering milk which was not attributable to the entrapment of the current deposit on vegetation came from the cumulative total in the soil, whereas it was evident from agricultural considerations that the direct entrapment of ^{90}Sr on vegetation in the previous year must make an appreciable contribution (the "lag-rate" effect). Second, the assumption that a constant fraction of the cumulative deposit in the soil enters plants each year was clearly incorrect because of the mechanisms (to which further reference is made later) which either remove it from the rooting zone or otherwise reduce its accessibility to plants.

Refinement to take account of these two defects was, however, impossible until more-extensive survey data were assembled. This was particularly true with respect to the second defect, since a reliable estimate of the manner in which p_d decreased with time could be expected only when ^{90}Sr that had been deposited in soil for many years contributed a major fraction of the contamination in milk. In the early years the direct contamination of vegetation was the dominant source of ^{90}Sr in diet. Accordingly, in long-term assessments it was at first necessary to assume a factor by which uptake from the soil decreased annually. The value of 2%, chosen by UNSCEAR in its first assessment of dose commitments from worldwide fallout,[4] was retained in the most recent assessment.[5] No factual justification for the use of this value has been advanced, however.

By the end of 1964, sufficient survey data existed to make possible some revision of Eq. 1. A marked lag effect of fallout in the previous year was implied by the fact that Eq. 1 led to an overestimate of the contamination of milk when the fallout at that time was low; the reverse was true when fallout was high (Fig. 1b). An improved but still empirical equation gave a significantly better fit to the data:[3]

$$C = p_r F_r + p_l F_l + p_d F_d \qquad (2)$$

The symbols are the same as those in Eq. 1, except that F_l represents the deposit in the last half of the previous year and p_l the lag-rate proportionality factor.

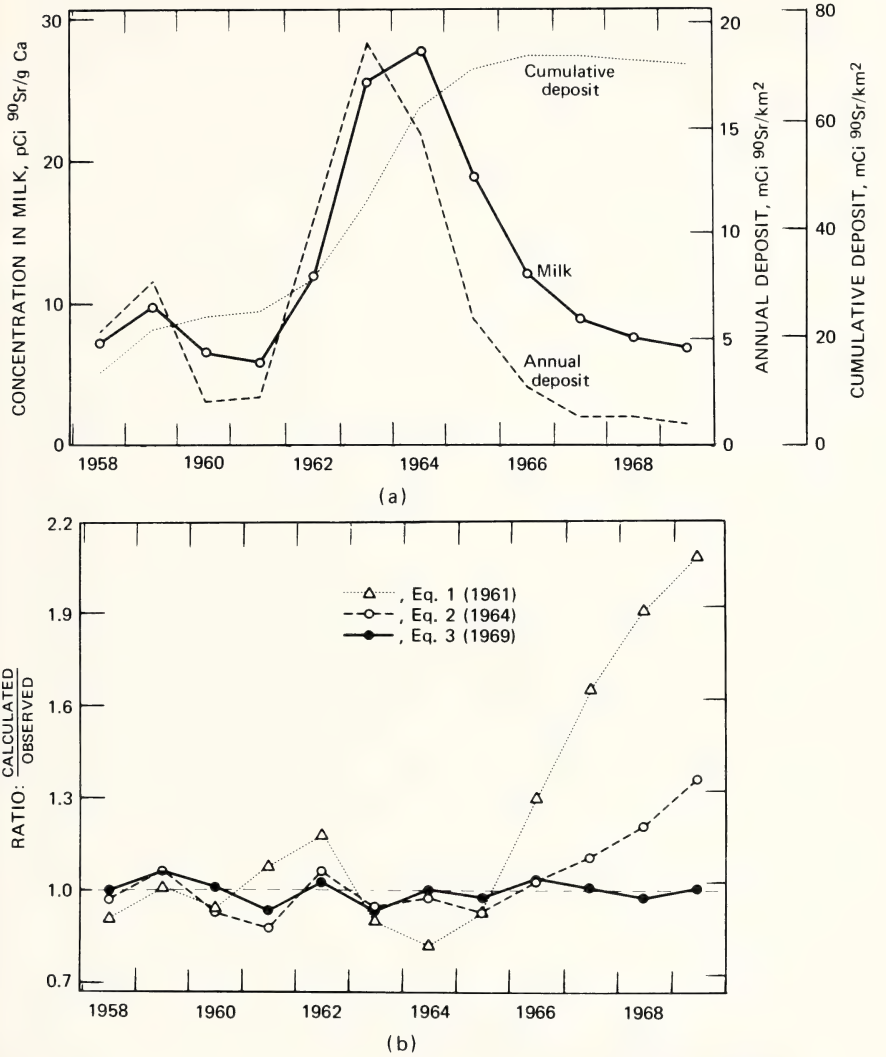

Fig. 1 Strontium-90 in fallout and milk in the United Kingdom, 1958 to 1969. (a) Mean results of surveys of deposition and contamination in milk conducted by the Atomic Energy Research Establishment, Harwell,[31] and Letcombe Laboratory,[32] respectively. (b) Annual average ratios of ^{90}Sr to calcium in milk calculated by alternative equations and expressed relative to observed values. Dates in parentheses indicate the most recent information available when the proportionality factors for the equation were derived.

This lag effect was found to be more closely related to the deposit in the last half of the previous year than to that in the whole year or in the summer months only. The following values for the proportionality factors were derived from survey data up to 1964: $p_r = 0.70$, $p_l = 1.13$, and $p_d = 0.11$. The annual average milk levels calculated in this manner not only agreed with those observed within 8% but also remained in equally good agreement in the two subsequent years (Fig. 1b), i.e., until 1966. Thereafter, however, when the rate of fallout was low and the cumulative deposit became the dominant source of contamination, the concentration in milk was consistently and increasingly overestimated. This defect was still more obvious with Eq. 1.

Therefore, as anticipated, p_d was apparently decreasing with time after the entry of ^{90}Sr into the soil. Revised calculations by both equations with the use of survey data up to 1969 gave lower values for p_d than had been derived when results for the earlier years only were available. However, the appropriate procedure was clearly to expand the third term of Eq. 2 to take account of the progressive reduction of p_d with time after deposition. The data being insufficient to permit the estimation of independent values for each preceding year, an exponential decrease in uptake from the soil was assumed:[6]

$$C = p_1 F_1 + p_2 F_{2b} + p_3 (s^{1.25} F_{2a} + s^2 F_3 + s^3 F_4 \ldots) \qquad (3)$$

where C = annual ratio of ^{90}Sr to calcium in milk (pCi ^{90}Sr/g Ca) in the current year (here designated year 1)

$F_1, F_2, F_3, \ldots$ = deposits of ^{90}Sr in year 1 and each previous year after correction for decay to midpoint of year (mCi/km^2)

a, b = first and second halves of year 2, respectively

s = reduction factor by which the uptake of ^{90}Sr from soil decreases annually through processes other than the decay of radioactivity

p_1, p_2, p_3 = proportionality factors

The first two terms on the right-hand side of the equation are similar to those in Eq. 2 but are not identical since they reflect the total effect of fallout, including the small contribution of uptake from the soil at the times in question, whereas in Eq. 2 uptake from the soil throughout the entire period is included in the third term. Simplifying Eq. 3 by considering F_2 as a whole was attempted, but a poorer fit to the data was obtained. This is in accord with the observation in the derivation of Eq. 2 that the lag-rate factor operated predominantly in the second half of the previous year.

The values of the coefficients in Eq. 3 derived from survey data up to 1969 are $p_1 = 0.70$, $p_2 = 1.41$, $p_3 = 0.20$, and $s = 0.86$. As shown in Fig. 1b, the content of ^{90}Sr in milk calculated on this basis agreed reasonably with that observed for each year between 1958 and 1969. Though still empirical, Eq. 3

describes the relation between the deposition of fallout and the contamination of milk considerably better than Eqs. 1 and 2; further improvement must await the availability of survey data for a longer period.

From the viewpoint of predicting dietary contamination over long periods, the particular advantage of Eq. 3 is that it provides an objective basis for estimating the extent to which the uptake of ^{90}Sr from the soil changes with time and thus dispenses with the need to make arbitrary assumptions. The value of 0.86 for s indicates a decrease by some 14% annually after allowance has been made for the decay of radioactivity. This value is in surprising agreement with the findings of Van der Stricht et al.,[7] who, applying a different type of analysis to survey results from Ispra in nothern Italy, deduced an annual reduction in uptake from the soil by about 13%. These values are considerably higher than 2%, the value assumed by UNSCEAR,[4] but it had long been evident that in some circumstances 2% was a gross underestimate. United Kingdom experiments showed that pasture grasses can remove 2 to 5% of recently introduced ^{90}Sr from soil in a single summer.[8] Beyond this the downward movement of ^{90}Sr in the soil by only a few centimeters will frequently cause an appreciable reduction in absorption since the roots of pasture plants draw nutrients largely from the upper soil layers.[9] Strontium-90, like calcium, can be leached to greater depths in the soil, and in some soils physicochemical changes may bring about a small reduction in uptake by plants.[10] All these processes operate conjointly, and the value of s now derived does not appear to conflict with any known facts. Note that, in addition to demonstrating a more rapid decrease in uptake of ^{90}Sr from the soil, Eq. 3 also indicates that absorption from this source is initially appreciably higher than was previously inferred. The value for p_3 is 0.20, whereas according to Eq. 2 p_d was estimated to be 0.11.

The limitations of the present analysis should be recognized, however. The time of year when fallout descends is likely to have an appreciable effect, especially in the first year, and no account can yet be taken of this fact. Furthermore, although exponential decrease in uptake from the soil has been assumed, there may be appreciable and as yet undetected changes in s with time; in particular, it is possible that this rate of change in the uptake of ^{90}Sr will slow down when ^{90}Sr has been present in soil for a longer period. Nonetheless, since Eq. 3 describes closely the situations in 1968 and 1969 (see Fig. 1), when the mean interval since the deposition of ^{90}Sr was 6 to 7 years, any eventual change in s should not have a large effect on the calculation of integrated doses.

Table 1 shows how improved calculations have modified estimates of the integrated total of ^{90}Sr that would enter milk in the United Kingdom from a given deposit. In the calculations using Eqs. 1 and 2 the UNSCEAR[5] value of 2% per annum decrease in uptake from the soil was assumed; no such assumption is required with Eq. 3. The earliest calculation (Eq. 1) appears to overestimate the integrated contamination of milk by a factor of about 2. This undoubtedly results largely from the assumption of only 2% annual reduction in uptake from

Table 1

ESTIMATES OF THE INTEGRATED TOTAL OF ^{90}Sr ENTERING MILK IN THE UNITED KINGDOM AFTER DEPOSITION OF 1 mCi ^{90}Sr/km^2

Method*	**Integrated contamination of milk, pCi ^{90}Sr year/g Ca**	**Fraction attributable to uptake from soil**
Equation 1 (1961)	4.9	0.84
Equation 2 (1964)	3.6	0.65
Equation 3 (1969)	2.3	0.47

*The dates in parentheses indicate the most recent data available when the calculation was made. With Eqs. 1 and 2, a 2% annual reduction in uptake from the soil is assumed following UNSCEAR;[5] no such assumption is needed with Eq. 3.

the soil; if a 5% reduction had been assumed, the integrated total derived by Eq. 1 would have been reduced by more than 30%. Therefore estimates of dose commitments from ^{90}Sr could have little quantitative validity until an objective basis was available for estimating the manner in which absorption from the soil would decrease with time. This comment implies no criticism of UNSCEAR for having assumed a considerably slower reduction in the uptake of ^{90}Sr from the soil than is now suggested. When information is lacking, the only safeguard against underestimating risk is to adopt cautious postulates, but the uncertainty they introduce must be remembered.

Unfortunately information on the transfer of ^{90}Sr to food chains, of the type provided by Eq. 3 for the United Kingdom, is not available for the majority of countries. Thus we must consider whether the relations derived for the United Kingdom by Eq. 3 are a reasonable guide to the general situation in other temperate regions. Here UNSCEAR[5] helps. Its tabulation of milk levels from 14 localities in the North Temperate Zone between 1955 and 1967 shows that the year-to-year trends in the United Kingdom were very close to the average (Fig. 2), the correlation coefficient being 0.99. The integrated total for the United Kingdom was about 10% higher (United Kingdom, 151 pCi year/g Ca; average, 137 pCi year/g Ca). Accordingly, for lack of better data, the United Kingdom relations for milk (derived by Eq. 3) will be assumed to be approximately representative of the average situation in other temperate countries until a more nearly complete assessment becomes available.

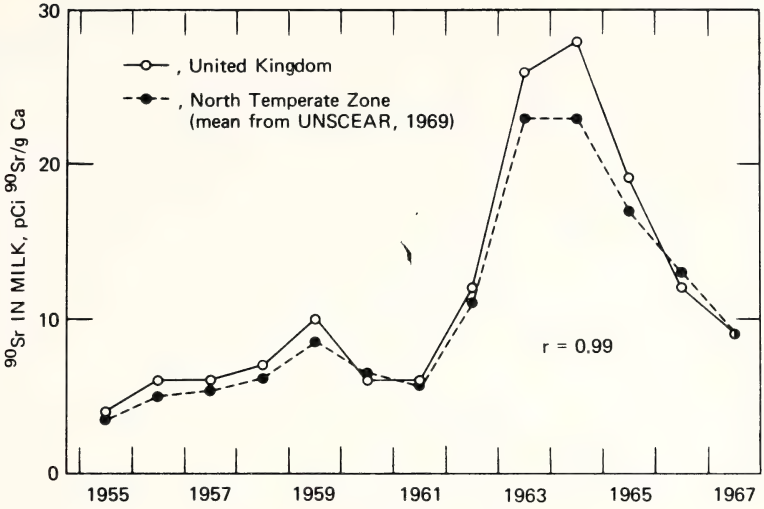

Fig. 2 Comparison of the annual average ratios of ^{90}Sr to calcium in milk in the United Kingdom with the mean values derived by UNSCEAR[5] for the North Temperate Zone.

RADIATION DOSES FROM LONG-LIVED FISSION PRODUCTS IN DIET AFTER A NUCLEAR WAR

Long-lived fission products from both the initial near-in deposit and the subsequent worldwide fallout will expose the survivors of a nuclear war to radiation. To illustrate problems that might arise, we will consider the situation in an area receiving an external radiation dose of 100 R/hr from early fallout 1 hr after the detonation of a weapon (the total amount of fission occurring in the entire war being 5000 Mt). From this model it is easy to scale upward or downward to any preferred case.

Attention is confined to doses received after sufficient time has elapsed for the contribution from short-lived fission products to be insignificant, for agricultural production to be resumed, and for dietary contamination from worldwide fallout to have reached its peak. For convenience, the 12-month period when this situation is attained is described as "postwar year 1," but we must realize that the length of time before this occurs could vary appreciably depending on many factors; it would not be likely to exceed 2 years, however. Since doses from long-lived fission products received before postwar year 1 would be small relative to the integrated total dose thereafter, little error is introduced by ignoring the earlier period.

Dose from ^{90}Sr

Assumptions on the composition of fission products, on the relation between deposition and external gamma radiation dose, and on fractionation, summarized in Appendix A, indicate that near-in fallout would deposit approximately 1000 mCi of ^{90}Sr per square kilometer in a fallout field of 100 R/hr at 1 hr. The large particle size of the debris will undoubtedly lower its solubility by a considerable factor, but, pessimistically, 500 mCi of ^{90}Sr per square kilometer is assumed to be present in forms accessible to plant roots in postwar year 1. The results of surveys of worldwide fallout combined with estimates of the quantity of nuclear fission released by nuclear tests, which are reviewed in Appendix B, suggest that 5000 Mt of fission would give rise to a deposit of about 1100 mCi of ^{90}Sr per square kilometer in the first year, with a half-residence time in the atmosphere of about 12 months; these estimates refer to temperate latitudes in the hemisphere where detonation occurred.

Applying the coefficients derived earlier for Eq. 3, we can derive the levels of ^{90}Sr in milk caused by the initial deposit and by worldwide fallout. These values, along with the fraction of the total contamination attributable to absorption from the soil in each year, are shown in Table 2.

Table 2

CONTAMINATION OF MILK WITH ^{90}Sr AFTER A NUCLEAR WAR*

	Contamination of milk, pCi ^{90}Sr/g Ca				
		Uptake from soil			
Years postwar	Direct contamination of plants with worldwide fallout	Worldwide fallout	Near-in deposit	Total	Fraction attributable to soil
1	1150	85	100	1340	0.14
2	570	200	85	860	0.33
3	290	230	72	590	0.52
4	140	230	60	430	0.67
5	72	210	50	330	0.78
6	36	180	42	260	0.86
7	18	160	35	210	0.91
8	9	130	30	170	0.95
9	4	110	25	140	0.97
10	2	95	21	120	0.98
Total					
Years 1 to 10	2290	1630	520	4450	0.48
Years 1 to ∞	2290	2130	630	5060	0.55

*For the calculations it is assumed that near-in fallout delivered 100 R/hr at 1 hr and that total fission in the hemisphere is 5000 Mt.

It is now widely recognized that, because of the risk of leukemia, the radiation dose to bone marrow is the appropriate basis for assessing risks from ^{90}Sr. To estimate the highest dose to this tissue which any individual could receive annually, we have assumed that the entire bone of infants in the first year of life is in equilibrium with diet each year, that the ratio of ^{90}Sr to calcium in their bone is 0.25 of that in the diet, and that 1 pCi ^{90}Sr/g Ca in bone will deliver 0.82 mrad/year to bone marrow.[11] On this basis, infants in their first year will receive the radiation doses shown in Fig. 3. In postwar year 1 the doses to bone marrow would be about 0.25 rad/year. Over the next few years the dose would decrease relatively rapidly to about 0.1 rad/year in the fourth year and about 0.03 rad/year after 10 years.

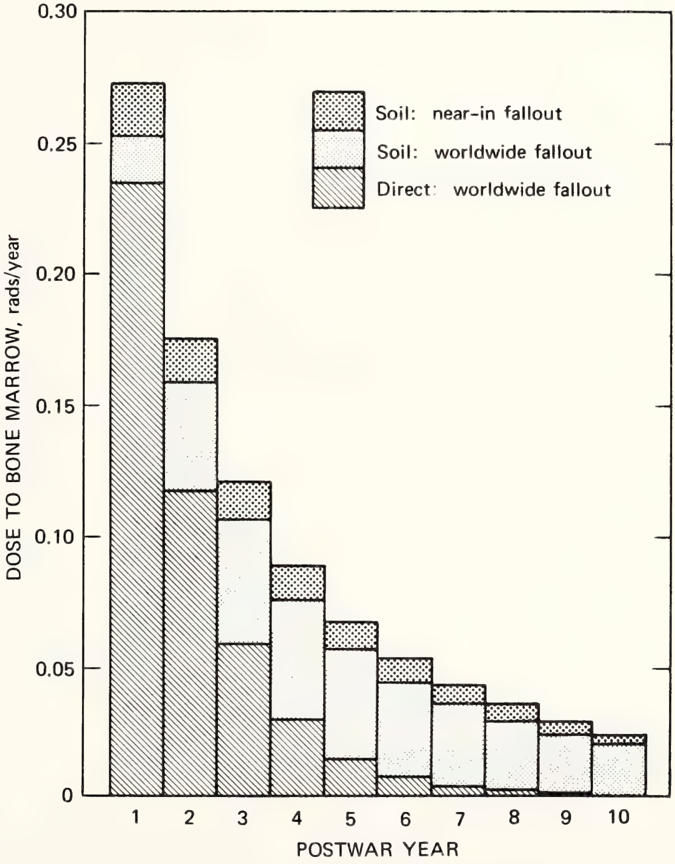

Fig. 3 Estimates of the radiation doses from ^{90}Sr to bone marrow which might be received in the first year of life by infants born during the decade after a nuclear war (for assumptions, see text).

Alternatively, the dose commitment to the population can be estimated by using the procedure of UNSCEAR.[5] In this calculation it is necessary to assume the relation between the ratio of ^{90}Sr to calcium in the total diet and that in milk. In the majority of countries where these ratios have been examined, the ratio in the total diet is 1 to 1.5 times that in milk. In the present calculation the higher (pessimistic) value of 1.5 was used. On this basis, the dose commitment from ^{90}Sr is about 1 rad, nearly all of which is received in the first 10 years (Table 3).

Table 3

DOSE COMMITMENT FROM ^{90}Sr TO BONE MARROW AFTER A NUCLEAR WAR*

	Dose to bone marrow, rads	
	Years postwar	
Source	**1 to 10**	**1 to ∞**
Worldwide fallout		
Direct contamination	0.38	0.38
Uptake from soil	0.27	0.35
Early fallout		
Uptake from soil	0.08	0.10
Total	0.73	0.83

*For the calculations it is assumed that near-in fallout delivered 100 R/hr at 1 hr and that total fission in the hemisphere is 5000 Mt.

Some 90% of the total dose commitment would come from worldwide fallout, the early fallout in an area where the external gamma dose was 100 R/hr at 1 hr contributing only a minor fraction of the total. This latter component could be scaled up to take account of situations in areas of much higher initial contamination. This would almost certainly be unrealistic, however, since the large particle size of the deposits in such areas would usually contain relatively insoluble fission products, whereas 50% solubility has been assumed in the present calculations. An appreciably larger contribution from this source might result, however, if the plumes from several weapons overlapped.

Dose from ^{137}Cs

So far we have considered doses from ^{90}Sr only. Cesium-137 must also be taken into account. Assessment of doses from this nuclide might be thought to be much simpler than that of doses from ^{90}Sr since, as is well known, the fixation of ^{137}Cs in clay minerals causes it to enter food chains to only a very small extent a year or two after deposition. Unfortunately, however, the basis for estimating doses from ^{137}Cs is less certain than that for ^{90}Sr, partly because, even though this discussion is concerned primarily with dietary contamination, it is logical to consider external as well as internal exposure from this nuclide and partly because its behavior cannot be related so precisely to that of a widely studied stable element as can that of strontium. However, the paucity of information regarding ^{137}Cs must be attributed largely to the fact that, when worldwide fallout first received notice, the isotopes of strontium were the dominant if not the sole preoccupation of many workers in this field. Such data as are available now have been reviewed by UNSCEAR.[5] The dose commitment to the bone marrow of the population from ^{137}Cs in worldwide fallout appears to be about 90% of that from ^{90}Sr, the same dose from ^{137}Cs being received by all tissues, of course. Within the limits of accuracy practicable in the present discussion, we may therefore assume that the dose commitment from ^{137}Cs to all tissues after the postulated war would be similar to that from ^{90}Sr to the bone marrow, i.e., about 1 rad.

The Total Dose

For present purposes it is unnecessary to consider nuclides other than ^{90}Sr and ^{137}Cs. Other fission products will be trivial sources of dietary contamination, and ^{14}C can be ignored because it would deliver considerably smaller annual doses in the decades following the war and because there is no prospect of influencing its transfer from the atmosphere into food chains.

Accordingly we may conclude that, after a nuclear war involving 5000 Mt of fission, the dose commitment from ^{90}Sr and ^{137}Cs to the inhabitants of the hemisphere in which the war took place would be approximately 2 rads to the bone marrow and 1 rad to the whole body from long-lived nuclides. For persons living near the target area, the doses would be only slightly higher than the average.

DISCUSSION

This assessment is, of course, approximate, but it may assist in a more realistic appraisal of the problems to which long-lived nuclides might give rise in the decades following a nuclear war. Even when the maximum allowance is made for uncertainties, the following facts are evident:

1. Doses from long-lived nuclides will be trivial relative to those received from short-lived activities in the earlier period in areas of appreciable near-in fallout.

2. Assuming that a nuclear war is of considerable magnitude (5000 Mt is in this category), worldwide fallout and not near-in debris would usually be the dominant source of dietary contamination with long-lived nuclides.

3. The direct contamination of growing crops is likely to be responsible for about half the dietary contamination with ^{90}Sr.

This last conclusion should not cause surprise. It is now over a decade since unequivocal evidence became available[12] that in times of relatively high fallout the direct contamination of plants and not, as it was first suggested, absorption from the soil was the major route by which ^{90}Sr entered diet. Implicit also in the analyses that could be made at that time was the fact that the average extent to which ^{90}Sr would enter plants from the soil over a long period was likely to be overestimated, but by a factor that could not be suggested until investigations had continued for a much longer period. That stage has now been reached. If the soil were low in calcium, the ^{90}Sr contribution could be greater than is suggested here. However, in a survival situation the need to achieve the maximum food production would probably be the most cogent reason to remedy such situations. (If sufficient calcium is present in soil for good crop growth, uptake of ^{90}Sr from the soil should not be appreciably greater than the average.)

So far this discussion has been concerned with the first question posed at the beginning of this paper, namely, "What radiation doses would long-lived nuclides portend after a nuclear war?" Now we will turn to the second question: "Is it prudent and realistic to prepare plans for long-term remedial action?" The literature contains numerous suggestions for modifying the transfer of fission products through food chains, but unfortunately the majority of them do not relate to situations likely to arise in practice.

A quarter or more of the casualties from long-lived nuclides after a nuclear war would apparently be due to external radiation from ^{137}Cs; this risk could not be mitigated over a wide area by any practicable method. Reduction of the average level of radioactivity in agricultural produce by a large factor also seems impossible. Figure 3 shows that, during the early years when doses would be highest, the major part of the internal dose from ^{90}Sr and, of course, almost the entire internal dose from ^{137}Cs comes from entrapment of the deposit on growing plants. Under normal agricultural conditions, reducing direct contamination of crops without destroying them would be impossible. Since we are unable to prevent either nuclide from entering the food chain, can we do anything to reduce transfer to man? The decontamination of milk has been widely discussed. Since milk products in all forms make a large contribution to the total contamination of diet, in round terms about half the dose commitment from ^{90}Sr and less than half the internal dose from ^{137}Cs could be spared by decontamination of the total milk supply. At one time it was imagined that this procedure would give greater protection to infants, especially from ^{90}Sr, than to adults, but, when it was recognized that ^{90}Sr is rapidly eliminated from the bones of the young,[13] it became evident that the benefit was much smaller.

Some other possible remedial measures have been suggested. When diets are low in calcium, the addition of that element reduces the retention of ^{90}Sr in the body, but increasing the calcium intake above that common in many western diets gives little benefit. Various therapeutic treatments have been discussed, but, since the risks from the therapy may be comparable to those from the anticipated radiation doses, the treatments cannot be considered seriously. Modification of the composition of diet, which has been suggested, would, in general, have no great effect if conventional methods of agricultural production were retained. Unfortunately the discrimination against strontium relative to calcium in passage from the diet of cattle to milk is offset by the greater direct contamination of the herbage cattle graze. Avoidance of foods that accumulate ^{137}Cs would seem equally impracticable. Therefore it seems that the intake of radioactivity in diet could be reduced by a considerable factor only if stocks of stored foods were available for several years or if crops were grown in greenhouses to protect them from direct contamination by fallout.

The following conclusions are inescapable: A large part of the dose from long-lived nuclides could not be avoided, and procedures available for mitigating some fraction of the dose would involve considerable effort and would possibly restrict food supplies.

Would it be reasonable to place this burden on the surviving population? In other words, would it be likely that casualties from radiation could be reduced enough to make the expenditure of effort worthwhile? This final question can be considered in two ways; the expected dose from long-lived nuclides can be compared with that to which the community is inescapably committed from natural background, or casualties to which long-lived nuclides would give rise in the absence of remedial action can be estimated.

Since the average natural background is about 0.1 rad/year, the dose commitment from long-lived fission products to the survivors of a war involving 5000 Mt of fission should be less than one-third of that received from background in the average life-span of man and considerably lower than that received in areas of high natural background. One could scarcely blame survivors of a nuclear holocaust if they felt that this risk was not worthy of consideration. However, to satisfy ourselves further, we will consider the number of casualties that might occur. An International Commission on Radiological Protection (ICRP) report[14] suggested that 1 rad delivered to one million persons might cause about 20 cases of leukemia and about 20 cases of other fatal cancers, the majority of which would not be in bone. On this basis we could expect about 40 leukemias per million of the population from the estimated total of 2 rads to bone marrow and about 20 cancers in other tissues receiving about 1 rad from ^{137}Cs only—a total of about 60 cases per million people. However, since a more recent assessment[15] suggests that these figures may be underestimated because of insufficient information on the length of the latent period, we will assume for prudence that up to 200 people per million might eventually die from cancer induced by the long-term components of fallout. The figure, of course, becomes more alarming when applied to a large population. Among 200 million persons, approximately the population of the United States, there might be 40,000 casualties during the recovery period. Although this is a large number, it is smaller than the number of annual fatalities on the roads of this country and of many other countries that are called advanced. In short, the total deaths caused by long-lived nuclides seem broadly comparable to the annual traffic death rate. Without expressing an opinion on the correctness of the community's attitude toward road safety, we would point out that road casualties could be greatly restricted by action that would impose a vastly smaller load on the resources of the community than would any measures to reduce casualties from long-lived nuclides after a nuclear war. Thus, by the standards the community now accepts, remedial action against the risks from long-lived nuclides would not seem justified; the number of casualties would be so small relative to the total loss and the difficulty of avoiding them would be so great that remedial action could not reasonably be contemplated.

We may conclude therefore that, in so far as our responsibilities lie in the field of civil defense, efforts to mitigate doses from radiation should be devoted solely to the early period when short-lived nuclides predominate. That is a sufficient problem.

APPENDIX A: DEPOSITION OF ^{90}Sr IN NEAR-IN FALLOUT WHEN EXTERNAL GAMMA DOSE IS 100 R/HR AT 1 HR

Dunning and Hilcken[16] estimated that a deposition of 800 MCi of mixed fission products per square mile 1 hr after fission would give an external gamma dose rate of 4000 R/hr at 3 ft above a theoretically flat plane. Assuming that the roughness of the ground would attenuate the external radiation dose by a factor of 2, that ^{90}Sr contributes 0.0013% of the total fallout activity at 24 hr[17] (adjusted for a half-life of 28 years), that mixed fission products are deposited in fission yield, and that they decay by a factor of 36 in 24 hr, the expected deposit of ^{90}Sr would be 5000 mCi/km^2 when the external gamma dose rate is 100 R/hr at 1 hr. An alternative calculation based on Glasstone[18] gives about one-third of this value, but we used the higher figure here, having in mind possible variability in different circumstances. We must, however, take account of the fractionation of fission products; the volatility of ^{90}Kr, the gaseous precursor of ^{90}Sr, is likely to deplete ^{90}Sr in the near-in deposit by a factor that may be conservatively estimated[19-22] at 5. Thus a deposit of 1000 mCi of ^{90}Sr per square kilometer is expected when the external gamma dose rate is 100 R/hr at 1 hr. There is much evidence[22-24] that the deposit in such areas will be of low solubility (probably not more than 10%), but, to avoid understatement of the quantities of ^{90}Sr which may enter food chains in subsequent years, we assumed 50% becomes soluble in the soil.

APPENDIX B: RELATION BETWEEN THE EXTENT OF NUCLEAR FISSION AND WORLDWIDE FALLOUT IN THE SAME HEMISPHERE

The pattern of fallout from the series of nuclear tests in 1962 provides a basis for estimating the deposition of ^{90}Sr in worldwide fallout after a nuclear war. Tests in the USSR are estimated to have yielded 60 Mt of fission[25] with a mean time of origin[26] at mid-September 1962. The average deposition of ^{90}Sr in the United Kingdom in 1963 was 19 mCi/km^2, and measurements of fission-product ratios[27] indicated that, in the spring and summer of that year, 70% of the ^{90}Sr was from tests held in 1962. This includes a small contribution from the United States 1962 equatorial tests. If we assume that 60 Mt of fission caused $0.7 \times 19 = 13.3$ mCi ^{90}Sr/km^2 to be deposited in the year after the detonations occurred, then, assuming similar latitude and height of injection, 5000 Mt of fission would give rise to 1100 mCi of ^{90}Sr per square kilometer in the first year after detonation.

The deposition in subsequent years would decrease at a rate depending on the residence time of the debris in the stratosphere and on interhemispheric transfer. From 1963 to 1966 the total content of the atmosphere decreased at a fairly steady rate, corresponding to a half-time for deposition estimated at 10 to 13 months; estimates of the longer half-time for interhemispheric transfer lie between 1.5 and 3.5 years.[28-30] For present purposes a round figure of 1 year has been taken as the effective half-time for the transfer of ^{90}Sr from the stratosphere onto the earth's surface.

REFERENCES

1. R. S. Russell, Dietary Contamination—Its Significance in an Emergency, in *Radiological Protection of the Public in a Nuclear Mass Disaster,* pp. 279–306, Fachverband für Strahlenschutz, Interlaken, 1968.
2. United Nations, Report of the Scientific Committee on the Effects of Atomic Radiation, General Assembly Official Records, Thirteenth Session, Supplement No. 17 (A/3838), New York, 1958.
3. B. O. Bartlett and R. S. Russell, Prediction of Future Levels of Long-Lived Fission Products in Milk, *Nature,* **209**: 1062–1065 (1966).
4. United Nations, Report of the Scientific Committee on the Effects of Atomic Radiation, General Assembly Official Records, Seventeenth Session, Supplement No. 16 (A/5216), New York, 1962.
5. United Nations, Report of the Scientific Committee on the Effects of Atomic Radiation, General Assembly Official Records, Twenty-fourth Session, Supplement No. 13 (A/7613), New York, 1969.
6. B. O. Bartlett, An Improved Relationship Between the Deposition of Strontium-90 and the Contamination of Milk in the United Kingdom, in Agricultural Research Council, Letcombe Laboratory, Annual Report 1970, p. 27, 1971.

7. E. Van der Stricht, P. Gaglione, and M. de Bortoli, Predictions of Strontium-90 Levels in Milk on the Basis of Deposition Values, Abstract of paper given at 2nd International Congress of the International Radiation Protection Association, May 3–8, 1970, Brighton, England, *Health Phys.,* **19**: 119 (1970). See also C. Stievenart and E. Van der Stricht. L'Évolution de la radioactivité ambiante au cours des années 1962 a 1966 et ses conséquences pour la contamination radioactive de la chaîne alimentaire, Euratom Report EUR4212f, 1968.
8. G. M. Milbourn, The Uptake of Radioactive Strontium by Crops Under Field Conditions in the United Kingdom, *J. Agr. Sci.,* **55**: 273–282 (1960).
9. P. Newbould, The Absorption of Nutrients by Plants from Different Zones of the Soil, in *Ecological Aspects of Mineral Nutrition of Plants,* I. H. Rorison (Ed.), pp. 177-190, Blackwell Scientific Publications Ltd., Oxford, 1969.
10. H. M. Squire, Long-Term Studies of Strontium-90 in Soils and Pastures, *Radiat. Bot.,* **6**: 49–67 (1966).
11. Medical Research Council, The Assessment of the Possible Radiation Risks to the Population from Environmental Contamination, Her Majesty's Stationery Office, London, 1966.
12. R. S. Russell, Radioisotopes and Environmental Circumstances: The Passage of Fission Products Through Food Chains, in *Radioisotopes in the Biosphere,* R. S. Caldecott and L. A. Snyder (Eds.), pp. 269–292, University of Minnesota Press, Minneapolis, 1960.
13. W. Fletcher, J. F. Loutit and D. G. Papworth, Interpretation of Levels of Strontium-90 in Human Bone, *Brit. Med. J.,* ii: 1225–1230 (1966).
14. International Commission on Radiological Protection, The Evaluation of Risks from Radiation, ICRP Publication 8, Pergamon Press, Inc., Oxford, 1966.
15. International Commission on Radiological Protection, Radiosensitivity and Spatial Distribution of Dose, ICRP Publication 14, Pergamon Press, Inc., Oxford, 1969.
16. G. M. Dunning and J. A. Hilcken (Eds.), The Shorter-Term Biological Hazards of a Fallout Field, USAEC Report M-6637, Division of Biology and Medicine and Armed Forces Special Weapons Project, December 1956.
17. R. C. Bolles and N. E. Ballou, Calculated Activities and Abundances of ^{235}U Fission Products, USAEC Report USNRDL-456, Naval Radiological Defense Laboratory, Aug. 30, 1956.
18. S. Glasstone, The Effects of Nuclear Weapons, USAEC Report ACCESS-127, Defense Atomic Support Agency, 1962.
19. G. M. Dunning, Biological Effects of a Nuclear Attack, USAEC Report TID-5563, Division of Biology and Medicine, September 1959.
20. K. Edvarson, K. Löw, and J. Sisefsky, Fractionation Phenomena in Nuclear Weapons Debris, *Nature,* **184**: 1771–1774 (1959).
21. E. C. Freiling, Radionuclide Fractionation in Bomb Debris, *Science,* **133**: 1991–1998 (1961).
22. J. F. Loutit and R. S. Russell, The Entry of Fission Products into Food Chains, *Progress in Nuclear Energy,* Series VI, Vol. 3, Pergamon Press Inc., New York, 1961.
23. E. A. Schuert, A Fallout Forecasting Technique with Results Obtained at the Eniwetok Proving Ground, in *The Nature of Radioactive Fallout and Its Effects on Man,* pp. 280–321, U. S. Congress Hearings, 1957.
24. T. Triffet, Basic Properties and Effects of Fallout, in *Biological and Environmental Effects of Nuclear War,* pp. 61–100, U. S. Congress Hearings, 1959.
25. Federal Radiation Council, Estimates and Evaluation of Fallout in the United States from Nuclear Weapons Testing Conducted Through 1962, F.R.C. Report No. 4, Superintendent of Documents, U. S. Government Printing Office, Washington, D. C., May 1963.

26. R. S. Cambray, E. M. R. Fisher, G. S. Spicer, C. G. Wallace, and T. J. Webber, Radioactive Fallout in Air and Rain: Results to the Middle of 1963, British Report AERE-R-4392, Nov. 18, 1963.
27. R. S. Cambray, E. M. R. Fisher, G. S. Spicer, C. G. Wallace, and T. J. Webber, Radioactive Fallout in Air and Rain: Results to the Middle of 1964, British Report AERE-R-4687, November 1964; and R. S. Cambray, personal communication.
28. D. H. Peirson and R. S. Cambray, Interhemispheric Transfer of Debris from Nuclear Explosions Using a Simple Atmospheric Model, *Nature,* **216**: 755–758 (1967).
29. P. Fabian, W. F. Libby and C. E. Palmer, Stratospheric Residence Time and Interhemispheric Mixing of Strontium-90 from Fallout in Rain *J. Geophys. Res.,* **73**: 3611–3616 (1968).
30. P. W. Krey and B. Krajewski, HASL Model of Atmospheric Transport, USAEC Report HASL-215, New York Operations Office (AEC), September 1969.
31. R. S. Cambray, E. M. R. Fisher, W. L. Brooks, and D. H. Peirson, Radioactive Fallout in Air and Rain: Results to the Middle of 1969, British Report AERE-R-6212, November 1969; and R. S. Cambray, personal communication.
32. Agricultural Research Council, Letcombe Laboratory, Annual Report 1969, Report ARCRL-20, 1970.

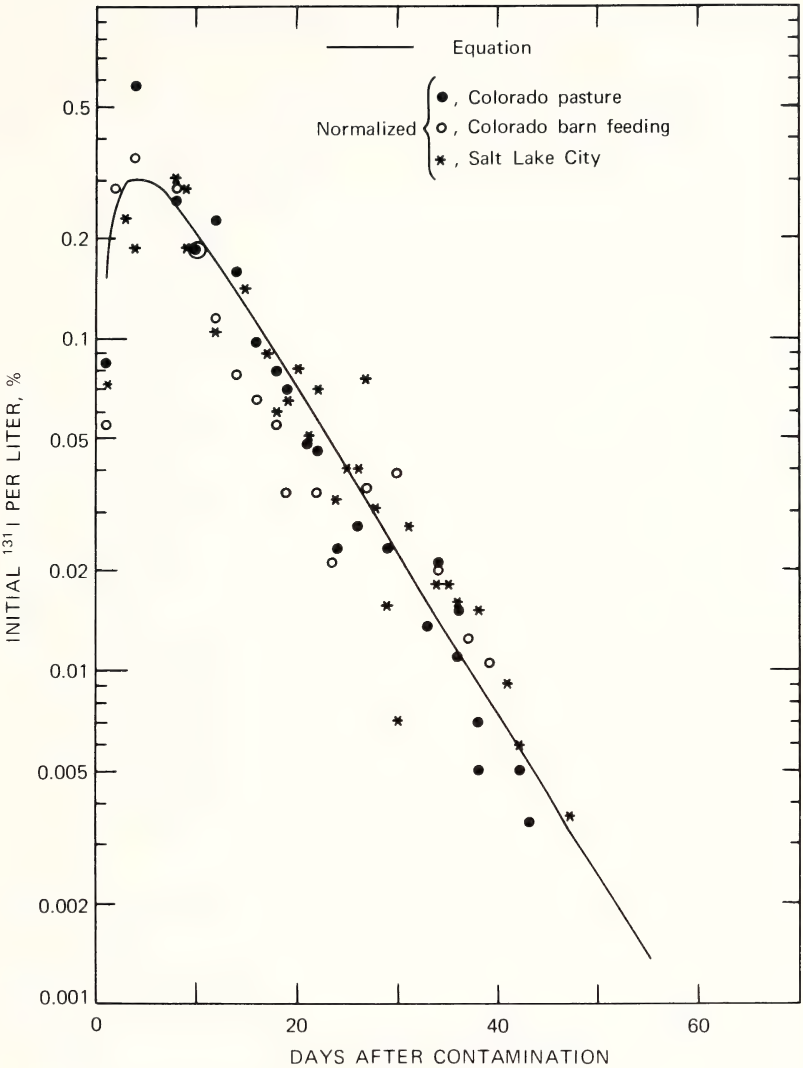

Fig. 1 Experimentally derived curve showing relation between ^{131}I levels in milk (%/liter) and time after contamination.

becomes progressively smaller. Action must be taken within about 4 days if the effectiveness is to be of the order of 90% (Refs. 6, 10, and 11). These procedures will involve various logistic problems of replacement of fresh milk by stored or processed milk and replacement of cattle feed by stored rations.

The same model can be used to predict the levels of radiocontamination when cows are returned to pasture or fed contaminated forage. As depicted in

UNITED KINGDOM CONSIDERATIONS IN AGRICULTURAL DEFENSE PLANNING

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ABSTRACT

The food-supply situation in the United Kingdom for 2 to 3 years postattack is examined. In the absence of imports and assuming a surviving population of 40 million (i.e., about 75% survival), a severe food shortage would exist.

Attention is drawn to the severe limitation on accurate assessment owing to a lack of knowledge about the beta-radiation contribution to the total radiation damage to crops and livestock from fallout.

In this paper the situation that would face the United Kingdom following a nuclear attack is set forth as objectively as possible. The basic data on which these conclusions are reached, such as the possible levels of contamination of foodstuffs and the effects of radiation on crops and livestock, are common to the situation of both the United States and the United Kingdom. There are, however, a number of other features that make the situation in the United Kingdom different from that in the United States, so much so that a different set of priorities would exist in the two countries.

THE PROBLEM

Any assessment of the effect of a nuclear attack on the United Kingdom's food resources over a 2- to 3-year period after an attack involves assumptions about the following factors:

1. The nature of the attack.
    - a. The number, distribution, and magnitude of the weapons burst.
    - b. Whether each weapon is airburst or ground burst.

   c. The meteorological conditions prevailing at the time of attack, including particularly the wind force and wind direction at various heights above the ground surface.
2. The size of the surviving population.
3. The size of any food stockpiles, particularly of foodstuffs that are normally widely dispersed throughout the country and thus relatively invulnerable.
4. The extent to which dispersion of food stocks and other ameliorative measures, such as putting animals under cover, can be effected.
5. The effect of fallout radiation on livestock and crops.
6. The magnitude and time of recommencement of imports of food.

Let us consider these factors in more detail.

The Attack

The war-games enthusiasts can devise a number of different attack patterns ranging widely in severity and especially in the number of ground-burst weapons involved. A typical attack pattern I have examined resulted in the situation defined in Fig. 1, which shows the area of the United Kingdom within which the radiation dose rate at D + 2 days exceeds a certain value. Such a graph gives no indication of the geographical distribution of the fallout pattern, but this is not important in the context of this paper.

Surviving Population

Any assessment of the adequacy of food supplies must obviously be related to the size of the population to be fed. Estimates of surviving populations depend on attack patterns and many other factors. The more optimistic assessments suggest that 80% of the population will survive and the more pessimistic 50%. That is, the surviving population will be between 40 and 25 million, the higher figure being the more likely.

Existing Food Stocks

In peacetime the United Kingdom imports about 50% of the food it consumes. Much of this imported food is stored and processed either in the area immediately around the big ports or in the large cities. Distribution tends to take place directly from these areas. Intermediate depots hold only small supplies, probably sufficient for a week or 10 days, and rely on rapid and efficient transport to maintain their stocks.

The total stocks in the country at any time depend on a variety of factors and differ for different commodities, but for the basic staples, i.e., mainly wheat and meat, supplies at normal rates of consumption would last for little more than 2 months. If an attack came without warning and no steps were taken to

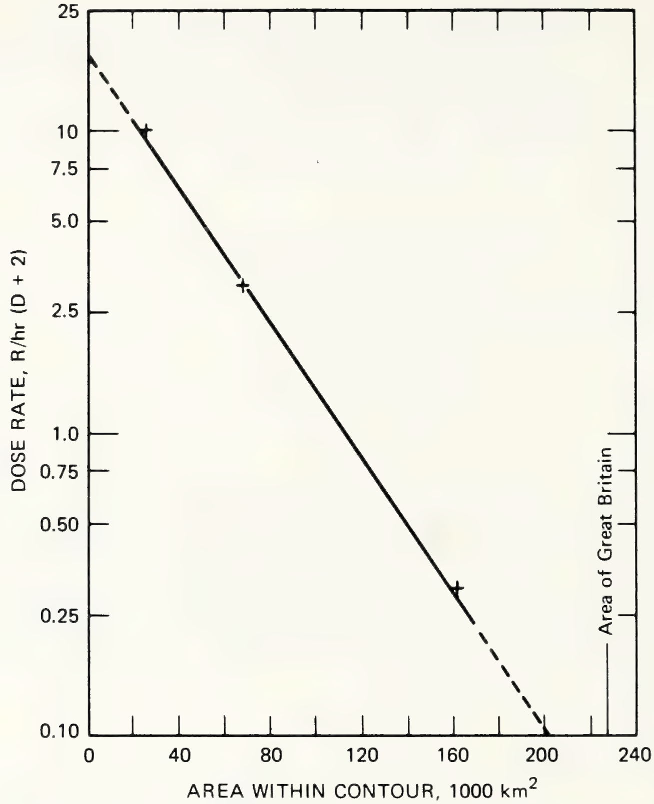

Fig. 1 **Area of Great Britain within which the radiation dose rate exceeds a specified value.**

conserve these food stocks, the bulk of the stockpile of food would be destroyed since the foodstuffs would undoubtedly be in target areas.

It is obviously desirable, therefore, to achieve dispersal of as much of these stocks as possible so that the probability of complete destruction is greatly reduced. Of course, the ideal situation would be for all the food available to be dispersed into every household in the land, and, although it is hoped that households would have on hand about a 2-weeks supply of food to tide themselves over the immediate postattack period, there are problems in achieving this desirable state of affairs.

Depending on the success of dispersal arrangements, more or less food would be available for the population. Most assessments suggest, however, that a greater proportion of food than of the population would be destroyed, so that in the end we should probably find that we had available perhaps between 1- and 2-months supply of food at normal rates of consumption.

Crops in Store

Until the next harvest we would have to rely on such crops as were in store and had survived the initial attack. In terms of calories these would be predominantly grains stored on farms and potatoes. About 10% of the grains are used for flour manufacture, 10% for distilling and brewing, and the rest for animal feed. Because these commodities are so widely dispersed, it is not expected that any appreciable proportion would be destroyed. I have assumed here that all such stocks would survive. Quantities available would vary from season to season. Table 1 shows the stocks of wheat and barley on hand at the end of each month from September to June along with the tons of cereal required to provide each member of a population of 40 million with 2000 Cal per day to the end of the following August. Note that, if the attack came after about the end of February, the existing grain stocks would be inadequate, even assuming that the barley available could be transformed into an edible and acceptable item of staple diet, such as a form of bread. Oat stocks are trivial and could make no significant contribution. Potato stocks are very variable since they depend on the potato market from year to year. In any event, it seems unlikely that potato stocks could provide more than 400 Cal per person per day, and in some years the contribution would be negligible.

Table 1

COMPARISON OF CEREAL STOCKS WITH REQUIREMENTS TO PROVIDE 40 MILLION SURVIVORS 2000 Cal PER PERSON PER DAY

Month	Cereal required, million tons	Wheat on hand, million tons	Barley required, million tons	Barley on hand, million tons	Oats required, million tons	Oats on hand, million tons
September	7.2	2.9	4.3	6.0		
October	6.6	2.5	4.1	6.2		
November	6.0	2.2	3.8	5.2		
December	5.2	2.0	3.2	4.5		
January	4.6	1.6	3.0	3.5		
February	4.0	1.2	2.8	2.6		
March	3.2	0.9	2.3	1.7	0.5	0.3
April	2.6	0.6	2.0	1.1	0.9	0.2
May	2.0	0.3	1.7	0.6	1.1	0.1
June	1.4	0.1	1.3	0.25	1.0	0.05

The situation that would arise should the number of survivors be 25 million is shown in Table 2. Here a difficult situation could occur if the attack came in May or June.

Table 2

COMPARISON OF CEREAL STOCKS WITH REQUIREMENTS TO PROVIDE 25 MILLION SURVIVORS 2000 Cal PER PERSON PER DAY

Month	Cereal required, million tons	Wheat on hand, million tons	Barley required, million tons	Barley on hand, million tons	Oats required, million tons	Oats on hand, million tons
September	4.5	2.9	1.6	6.0		
October	4.1	2.5	1.6	6.2		
November	3.7	2.2	1.5	5.2		
December	3.3	2.0	1.3	4.5		
January	2.9	1.6	1.3	3.5		
February	2.5	1.2	1.3	2.6		
March	2.0	0.9	1.1	1.7		
April	1.6	0.6	1.0	1.1		
May	1.3	0.3	1.0	0.6	0.4	0.1
June	0.9	0.1	0.8	0.25	0.55	0.05

Table 3

ESTIMATED FALLOUT-RADIATION LETHALITY ($LD_{50/60}$) FOR LIVESTOCK EXPOSED TO GAMMA RAYS*

Animal	In barns (gamma)	In pens or corrals (gamma + skin beta)	In pasture (gamma + skin beta + G.I. beta)
Cattle	500	450	180
Sheep	400	350	240
Pigs†	640	600	550

*According to Bell, Sasser, and West.[1]

†Pigs do not normally forage in the open.

Surviving stocks from other sources, e.g., in the hands of millers, would be small and would probably be no more than those needed to compensate for the small loss of farm stocks that would inevitably result from the attack.

Livestock

Livestock would be affected by radiation from fallout. Animals in open fields would be exposed not only to gamma radiation from fallout but also to irradiation of the skin from particles adhering to it and to beta irradiation of the gastrointestinal (G.I.) tract from ingested fallout material. Estimations by Bell, Sasser, and West[1] of the equivalent gamma-radiation dose corresponding to $LD_{50/60}$ for animals exposed to gamma radiation alone, to gamma + skin beta radiation, and to gamma + skin + G.I. beta radiation are shown in Table 3. Assuming that the livestock population is uniformly dispersed geographically, the population would be reduced by the proportion of the country receiving radiation in excess of the appropriate LD_{50} dose. (A reasonably accurate estimate of survivors is obtained by assuming that all animals receiving a dose greater than the LD_{50} die and that all others survive.) Using the data in Fig. 1 and Table 3, we can calculate the surviving animal population. The results are shown in Table 4, together with the effect of putting the animal population under cover for protection from the beta effects and for a gamma-radiation protection factor of 2 or 3.

Table 4

LIVESTOCK SURVIVORS IN MILLIONS

Animal	Preattack population	Gamma exposure only	Gamma + skin beta + G.I. beta exposure	In shelters (protection factor of 2)*	In shelters (protection factor of 3)*
Dairy cows	4.6	3.1	2.5	3.6	4.0
Beef cows	6.2	4.2	3.3	4.8	5.4
Pigs	6.8	4.9	4.7	5.8	6.3
Sheep (June)	29.0	18.6	16.0	22.0	24.0
Sheep (December)	20.0	12.8	11.0	15.0	16.4

*Livestock in shelters would receive gamma exposure only.

Pasture Requirement

In the United Kingdom a cow requires about 1 acre and a sheep $\frac{1}{5}$ acre of pasture to provide a maintenance diet for an adult animal or to provide for the growth of an immature animal. The pasture requirement will therefore range from 8.8 million acres for the lowest number of survivors shown in Table 4 to 14.4 million acres for the highest number. Currently the acreages of pasture in Great Britain are:

11.1 million acres of permanent grass
5.6 million acres of clover and rotational grass
17.0 million acres of rough grazing

These acreages can be expected to be reduced to 8.8, 4.5, and 13.6 acres, respectively, because of radiation damage, assuming that beta radiation doubles the effect of radiation dose. The amount of pasture available would therefore be adequate provided the additional effect of beta radiation did not increase the effective radiation dose by a factor of more than 2 or 3. The pig population, of course, since pigs are direct competitors with man for grain, would have to be rapidly reduced. While this was taking place, our main meat ration would be pork and bacon for a considerable time.

Meat Yield

The amount of meat obtained from the various forms of livestock considered is shown in Table 5. The figures in column 3 are derived from the reproduction rates obtainable in peacetime. Using the animal survival figures given in Table 4, we can calculate that about 1 million tons of meat per year would be available (more if the animal population were to be decreased). This could give a ration of about 1 lb of meat per week (equivalent to about 1000 Cal per week) to each person of a population of 40 million survivors.

Table 5

MEAT YIELD PER ANIMAL

Animal	Yield, lb/animal	Yield,* lb/year
Dairy cow	350	100†
Beef	200	100
Pig	40	360‡
Sheep (June)	60	60
Sheep (December)	90	90

*Figures in this column are derived from the reproduction rates obtainable in peacetime.

†Some slaughtering of the dairy herd for meat is assumed.

‡The maximum is 360 lb/year. If the pig population is to be reduced, 40 lb/animal would be the more appropriate value.

Growing Crops

With the exception of pasture crops, none of the crops growing in the field at the time of the attack would be of any value as food until they were harvested. For our present purposes attention will be confined to the staples—wheat, barley, potatoes, and pasture, with pasture including "grass" for grazing, hay, and silage production. Losses of these crops can result from the following:

1. Direct physical destruction.
2. Loss due to interference with normal agricultural practice, e.g., weeding, spraying, etc.
3. Radiation damage.

Losses due to direct physical destruction and interference with normal agricultural practice are likely to be slight and are ignored here. Radiation damage, on the other hand, could be severe for some crops. Data on the effect of gamma radiation are now plentiful, and detailed assessments could be made using these data and particular fallout patterns. Unfortunately such assessments of themselves would be largely meaningless since they would fail to take into account the effect of the beta-radiation component from fallout. Estimates of the additional effect of beta radiation vary and will certainly differ for different crop species and times of the year. What is important from our point of view is to decide whether a knowledge of the effect of the beta-radiation component is vital to our assessment. The following simple example indicates that the factor limiting the precision of any assessment is, in fact, the effect of the beta component, and until this is resolved little purpose will be served by further refinement in our assessment of the gamma-radiation effect. In this assessment I have assumed:

1. Uniform geographic distribution of each crop so that the damage is related to the area of the country under the relevant levels of radiation dose.
2. A single radiation-dose-effect relation for each crop.

The justifications for such sweeping assumptions are: First, our assessment of the area of the country under given radiation dose levels is extremely crude, because of, among other things, uncertainties in the time of arrival of the fallout, and, second, these assumptions simplify the demonstration that improvements in our knowledge of the effect of beta radiation are important if reliable assessments are to be made. Table 6, which lists the results calculated for wheat, barley, potatoes, and pasture, shows that we could expect in the United Kingdom to lose one-quarter to one-half of our cereal crops and up to one-quarter of our pasture. There would be severe retardation of growth of nearly one-half of the pasture, but potato crops would not be appreciably affected. Table 7 shows the relation between the expected yields of cereals at the next harvest and the requirement to give 2000 Cal per person per day to 25 and 40 million survivors. There is likely to be an overall deficiency of cereals, and this deficiency could be much greater if the beta radiation from fallout increased the effective radiation dose to the crop by a factor of 3 or 4.

Thus we can conclude that, before any reliable estimates can be made based on a regional analysis of the situation and specific attack patterns at particular times, we shall need to have a much clearer idea of the contribution that beta radiation from fallout makes to the total radiation dose received by crops.

CONCLUSION

The very crude assessments made in this paper suggest that, if an attack occurred any time between the early months of the year and the next harvest,

Table 6

RADIATION SENSITIVITY OF CROP PLANTS AND ANTICIPATED LOSS

Crop	Dose (D) resulting in negligible yield*	Dose rate at D + 2 to give D over the period H + 2 to H + 24	Area of country* receiving dose greater than D	Area assuming† beta doubles effective dose	Area assuming† beta quadruples effective dose
Wheat	>1000 R	> 6 R/hr	20%	30%	40%
Barley	> 500 R	> 3 R/hr	30%	40%	55%
Potatoes	>4000 R	>20 R/hr	Negligible‡	Negligible‡	20%
Pasture	>4000 R	>20 R/hr	Negligible‡	Negligible‡	20%
	>2000 R growth retarded for 1 month	>10 R/hr	Negligible‡	20%	40%

*When plant is mature and crop is ripening, there would be little effect on yield.

†Reduction in yield of crop is assumed equal to area of country affected.

‡An area less than 10%.

Table 7

CEREAL YIELDS AT NEXT HARVEST AND REQUIREMENTS OF SURVIVING POPULATION

	Grain yield, million tons			Cereal required to give 2000 Cal/day, million tons	
Crop	No loss	25% loss	50% loss	40 million survivors	25 million survivors
Wheat	3.4	2.6	1.7		
Barley	4.4	3.3	2.2		
Total	7.8	5.9	3.9	8.0	5.0

there would likely be a severe deficiency of calories, mainly because of the inadequate quantities of grain available. Meat, potatoes, and other foods would be unlikely to fill the gap. There would probably be a considerable but not a disastrous reduction in the animal population. The number of survivors could be appreciably increased if the animals could be put under cover, particularly under cover with a protection factor of 2 or 3. Sufficient pasture to support the surviving livestock would probably be available, and it is assumed that the pig population would be greatly reduced to conserve cereals for human consumption. The yield of crops standing in the field would probably be reduced by one-quarter to one-half of that expected, mainly because of radiation damage. It is unlikely that the final crop yield would be sufficient to provide adequate diet for the surviving population.

In all these assessments, for the size of attack envisaged in the model, the beta-radiation component of fallout clearly has an appreciable effect on the numerical results obtained. Although we could carry out a more refined exercise, taking into account actual distributions of livestock and crops and the fallout pattern from specific model attacks, it is doubtful whether much more meaningful assessments could be made until we have a clearer idea of the significance of the beta radiation from fallout. It is clear that, if the results of a nuclear attack were anything like those estimated in this assessment, there would be a chronic shortage of food over a period of at least 2 years. Stocks could be supplemented only by importing food. We must endeavor to refine our knowledge of the beta-radiation contribution to the total damaging effect of fallout since this will influence the importance we must attach to our ability to import foodstuffs. Similar assessments for other countries would indicate their ability to produce surpluses for export to the United Kingdom. We still have some way to go in the United Kingdom before we can make a satisfactory assessment of the medium-term situation that would confront us after a nuclear attack.

REFERENCE

1. M. C. Bell, L. B. Sasser, and J. L. West, Simulated-Fallout-Radiation Effects on Livestock, this volume.

Table 1

MEDIAN LETHAL DOSE (LD_{50}) VALUES FOR LARGE ANIMALS EXPOSED TO GAMMA OR X IRRADIATION*

			Median lethal dose (LD_{50})		
Radiation source	**Dose rate, R/min**	**Method of exposure**	**Midair dose, R**	**Mid-tissue dose, rads**	**Reference**
		Burros			
1000-kVp X ray	7.5	Bilateral	369	155	8
^{60}Co	0.85	Multisource	784	280	13
^{182}Ta	0.37	Multisource	641	290†	14
^{95}Zr–^{95}Nb	0.28	Multisource	385	350†	15
		Cattle			
^{60}Co	6.6	Multisource	200	125‡	16
^{60}Co	0.9	Multisource	450	150	7
^{60}Co	0.9	Multisource	543	160	7
		Goats			
2500-keV gamma	32.5	Bilateral	395	240‡	17
1000-kVp X ray	7.5	Bilateral	312	200	12
^{60}Co	1.3	Bilateral	550	350‡	18
		Sheep			
^{60}Co	11.0	Bilateral	237	145	5
1000-kVp X ray	7.5	Bilateral	252	146	5
1000-kVp X ray	7.5	Bilateral	314	189	11
250-kVp X ray	7.5	Bilateral	389	245	6
^{60}Co	4.35	Bilateral	318	194	9
^{60}Co	0.5	Bilateral	338	206	9
^{60}Co	0.3	Multisource	524	205§	19
^{60}Co	0.06	Free moving¶	495	302	9
^{60}Co	0.033	Free moving¶	637	389	9
		Swine			
^{60}Co	50.0	Bilateral	350 to 400	240	2
1000-kVp X ray	30.0	Bilateral	510	250**	4
^{60}Co	18 to 29	4 pi	393	228	20
^{60}Co	18 to 29	4 pi	335	218	20
1000-kVp X ray	27.0	Bilateral	425	255	21
2000-kVp X ray	15.0	Bilateral	350 to 400	230**	4
^{60}Co	11.5	Bilateral	375	260	22
^{60}Co	10.0	Bilateral	400 to 450	270	2
1000-kVp X ray	9 to 10	Bilateral	399	270	3
^{60}Co	1.0	Bilateral	650 to 700	425	2
^{60}Co	0.85	Multisource	618	370**	13
^{60}Co	0.067	Free moving¶	2000 to 2500	1350 to 1700	10

*Only studies in which a relatively homogeneous depth-dose distributions were obtained are presented in this table. Those in which unilateral or dorsal–ventral exposures or low-energy radiations were utilized are not included.

†Value is estimated by Trum.[15]

‡Estimate is based on data presented in the reference cited.

§Value is estimated by Bond.[1]

¶Although they were exposed from one direction, the animals' random-movement resulted in equal exposure to both sides, providing an effective bilateral exposure.

**Value is estimated by D. Brown.[2]

Table 2

SURVIVAL OF SHEEP AND GOATS EXPOSED CONTINUOUSLY TO ^{60}Co RADIATION

Species	Dose per day and dose rate	Mean survival time, days	Mean cumulative lethal dose, R
Sheep[23]	46 R at 0.033 R/min	43 (males)	1975
Goat[24]	40 R at 0.033 R/min	57 (males)	2280
		50 (females)	2000
	30 R at 0.025 R/min	85 (males)	2550
		81 (females)	2430
	15 R at 0.013 R/min	240 (males)	3600
		161 (females)	2415
	7.2 R at 0.007 R/min	1152 (males)	8330
		384 (females)	2650

Table 3

SURVIVAL OF LARGE ANIMALS EXPOSED TO FRACTIONATED DAILY EXPOSURES OF ^{60}Co RADIATION*

Species	Dose per day and dose rate†	Mean survival time, days	Mean cumulative lethal dose, R
Burro	Single dose	25	
	400 R at 0.28 R/min	8	3,320
	200 R at 0.14 R/min	14	2,820
	100 R at 0.07 R/min	23	2,330
	50 R at 0.035 R/min	30	1,510
	25 R at 0.017 R/min	63	1,575
Cattle	Single dose	20	
	100 R at 0.07 R/min	32	3,200
	50 R at 0.035 R/min	45	2,250
Swine	Single dose	15	
	100 R at 0.07 R/min	39	3,900
	50 R at 0.035 R/min	205	10,250

*These studies were conducted at the UT–AEC Agricultural Research Laboratory; the multiple-source (^{60}Co) exposure at a dose rate of 0.5 to 0.85 R/min was used (Brown[2]).

†Dose rate in roentgens per minute calculated as though the daily exposure was continuous for the entire 24 hr per day.

RADIATION EFFECTS ON FARM ANIMALS: A REVIEW

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ABSTRACT

Hematopoietic death would predominate in food-producing animals exposed to gamma radiation under fallout conditions leaving animal survivors. Gamma-radiation doses of about 900 R would be lethal to 50% of poultry, and about half this level would be lethal for cattle, sheep, and swine. Grazing cattle and sheep would suffer most from combined radiation effects of skin-beta and ingested-beta radioactivity plus the whole-body gamma effects. The $LD_{50/60}$ for combined effects in ruminants is estimated to be at a gamma exposure of around 200 R in an area where the forage retention is 7 to 9%.

Either external parasites or severe heat loss could be a problem in skin irradiated animals. Contrary to early reports, bacterial invasion of irradiated food-producing animals does not appear to be a major problem. Productivity of survivors of gamma radiation alone would not be affected, but, in an area of some lethality, the productivity of surviving grazing livestock would be severely reduced owing to anorexia and diarrhea. Sheltering animals and using stored feed as countermeasures during the first few days of livestock exposure provide much greater protection than shielding alone.

The purpose of this review is to summarize the data available on the effects of ionizing radiation on food-producing animals which would be of value in predicting the effects that could be encountered from radioactive fallout in the event of nuclear war. Most of the data are limited to somatic effects of gamma and beta radiation on survival and productivity of cattle, swine, and sheep. Although much more information is available on radiation effects in small laboratory animals, it is difficult to extrapolate these data to large food-producing animals exposed to a combination of internally and externally applied radiation. Some attention is also given to measures that could be used to reduce radiation exposure of food-producing animals.

Ionizing radiation from radioactive fallout occurs principally as beta particles and gamma rays. The median beta energies are between 0.3 and 0.4 MeV, but the maximum may be up to 5 MeV. Most of the data available on beta irradiation effects on food-producing animals were obtained by using either ^{90}Y or ^{90}Sr–^{90}Y, which have higher average energies than are characteristic of local fallout. Information on gamma irradiation was obtained principally by exposing large animals to ^{60}Co or ^{137}Cs, which have penetration characteristics similar to gamma fallout radiation.

Limited information is given on neutron exposures, and none is given on alpha radiation since neither of these emissions is expected to be of any consequence in radioactive-fallout effects on food-producing animals.

RADIATION LETHALITY

General

Exposures to gamma radiation at dose rates expected under fallout conditions causing early deaths in about half of the animals are expressed as a dose lethal to 50% in either 30 or 60 days ($LD_{50/30}$ or $LD_{50/60}$). This mortality level varies with dose rate, quality and type of radiation, animal species, and a number of other variables. The upper and lower limits of the distribution of radiation deaths for adult cattle, swine, and burros are shown by the typical sigmoid curves in Fig. 1. The data obtained from ^{60}Co exposure to

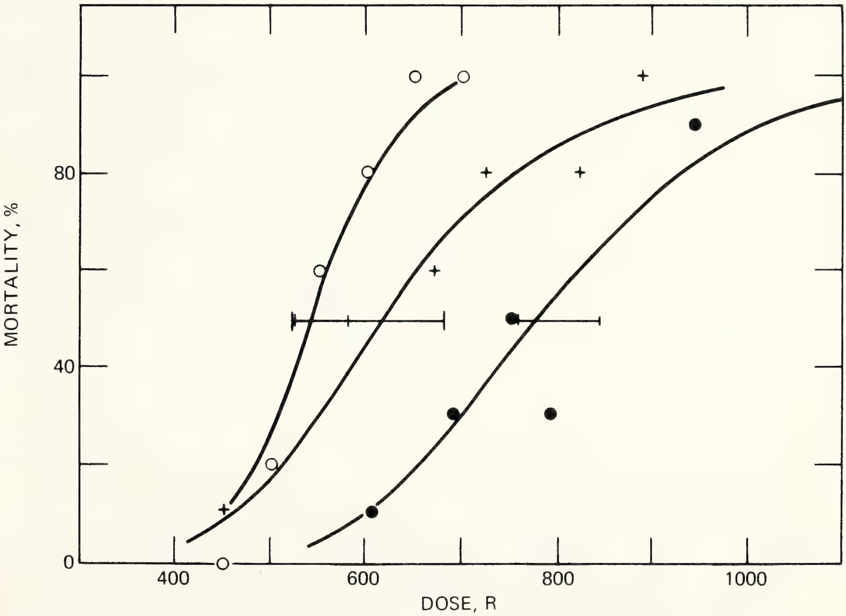

Fig. 1 Mortality of three species exposed to ^{60}Co at a dose rate between 0.5 and 1 R/min. ○, cattle; +, swine; ●, burros; ⊢⊣, 95% confidence interval. (Data from D. G. Brown, UT–AEC Agricultural Research Laboratory.)

dose rates of 0.5 to 1 R/min show the species variation among these large animals. This variation is much greater at the 99% mortality level than for 1% mortality.

The gamma-radiation dose levels are usually expressed as either midline "air dose" or midline absorbed dose as discussed by Page.[1] Because of tissue mass, the gamma-radiation midline dose from fallout would be reduced by at least 50% in adult cattle, but for poultry the reduction would be inconsequential. In this review the gamma-radiation exposures are listed as the air dose to the animals, and the units are in roentgens. The data most applicable for gamma radiation from fallout in which there would be at least 20% survival of continuously exposed animals would be in fallout-deposition areas where the dose rate would not be expected to exceed 2 R/min for a period of over 1 hr. An exception to this rule would be animals that might be moved from a heavily contaminated field into a protective shelter until the early fallout had decayed to a nonlethal level. A review by Page[1] showed that dose-rate effects are considerable in swine and very slight in sheep and burros. Sheep were more sensitive than swine at all dose rates reported. Reduction of the dose rate from 2 R/min to 0.1 R/min increased the estimated LD_{50} dose to sheep by 20% and to swine by 340%. Swine also show 50% recovery in 3 days from acute gamma-radiation exposure, whereas sheep require about 13 days.[2] In a review article Brown and Cragle[3] reported that the swine $LD_{50/30}$ at 0.85 R/min was 618 R but at 18 to 29 R/min only 310 R. They also reported that young cattle are more sensitive to gamma irradiation than adult cattle. In general, it is assumed that the young are more sensitive to radiation; however, Case and Simon[4] reported that at a dose rate of 4 R/min the $LD_{50/30}$ for newborn pigs was 375 R, which is near the estimated $LD_{50/30}$ for older swine. Data for predicting dose-rate effects in cattle are very limited, but, in general, the higher the dose rate, the lower the $LD_{50/30}$ is in the species studied (Fig. 1). Fallout dose rate varies with weapon yield, type of burst, distance downwind, wind speed, and number and frequency of detonations. Considering both fallout and animal species variables, it appears that the most useful data would be those obtained on animals continuously exposed to early fallout, but no such data were found; therefore only information on animals exposed to a gamma dose rate of from 0.1 to 2 R/min is considered.

Symptoms of Gamma-Fallout-Radiation Sickness

The primary symptoms expected from gamma radiation alone at levels to produce some deaths in farm animals are those associated with damage to the hematopoietic system. These usually include a severe drop in blood platelets to the point that blood would be lost into intracellular spaces and from both the respiratory and gastrointestinal (GI) tracts owing to failure in blood clotting. Increased capillary permeability also contributes to loss of blood cells, plasma, and electrolytes. Most of these losses occur between 14 and 30 days after exposure when there are also low white-cell counts, sometimes accompanied by pyrexia and bacterial invasion.[5] Cattle exposed to 200 to 600 R at dose rates of 0.5 to 1 R/min usually show mild anorexia and slight pyrexia for about 24 hr; they then appear normal until about 14 days, when there is a marked pyrexia in those lethally irradiated; the survivors show a mild pyrexia.

Anorexia and vomiting, which may be associated with the gastrointestinal death syndrome, would be expected in few if any of those surviving gamma irradiation. At the exposure and dose rate considered, the central nervous system (CNS) may be affected in some burros.[3] Data on burros are included since meat of equine origin is consumed at the annual rate of 2 to 3 kg per person in some European countries. The CNS and gastrointestinal death syndromes in gamma-irradiated animals would be expected only if the dose rate were higher than 2 R/min. Vomiting, anorexia, and weight loss were reported in swine exposed to 250 to 700 R of ^{60}Co gamma radiation at 21 R/min giving an approximate $LD_{50/30}$ of 335 R for 33-kg swine.[6] None of these symptoms were seen in 30-kg growing swine surviving an exposure to 450 R at 0.6 R/min from a multisource ^{60}Co field.[7] Pigs that died showed anorexia and blood loss for only 2 days prior to death, which occurred 16 to 20 days after exposure. Case and Simon[4] observed a mild transitory diarrhea in newborn pigs exposed to ^{60}Co and found an $LD_{50/30}$ of 375 R at 4 R/min. These pigs had diarrhea at 2 to 5 days after irradiation and cutaneous hemorrhages at 9 to 14 days; all deaths occurred between 10 and 29 days postirradiation. Therefore it appears to be very important to consider the dose rate in determining the symptoms and $LD_{50/30}$ in swine exposed to gamma radiation. These data would probably also apply to other species of food-producing animals, but available data are insufficient for definite conclusions.

Beta-Radiation Effects

Predictions by the National Academy of Sciences–National Research Council (NAS–NRC) committee,[8] based primarily on data gained from dosimeter readings in dogs and goats fed sublethal levels of $^{90}YCl_2$ solution,[9] were that the large intestine would be the critical organ and that fallout ingested by grazing livestock would be of little consequence compared with gamma-radiation effects. These conclusions were based on the assumption that fallout would be homogeneously mixed with the contents of the GI tract.

Although no research data are cited, a 1965 USSR textbook[10] entitled *Civil Defense in Rural Regions* states that inflammation of the mucosa of lips, gums, and the deep part of the oral cavity occurs in livestock consuming contaminated feed and water. These symptoms appear after 7 to 11 days, when the animals become lethargic and refuse to eat. They also noted considerable hair loss, and the further course of radiation illness depended on the degree of injury to internal organs. In 1967 Bell[11] reported that the omasum and rumen were the organs most severely affected in sheep given ^{144}Ce–^{144}Pr chloride solution in their feed. These data on sheep were obtained for levels at which 50% of the sheep developed diarrhea and half of those with diarrhea died.

More recently Bell et al.[12] showed that feeding an insoluble fallout simulant can be lethal to sheep and that it severely affects the productivity of survivors. The simulant consisted of ^{90}Y fused to 88- to 175-μ sand to provide about 10 mCi/g of sand. The primary symptoms from feeding 0.8 to 3.2 mCi/kg of body weight were anorexia, diarrhea, weight loss, and pyrexia. Sufficient radioactive sand had "pocketed" in areas of the rumen and abomasum to cause ulceration and fibrin infiltration of the mucosa. Readings from microdosimeters implanted in the "pockets" of the abomasum averaged eight times as high as those in the small and large intestine. No gross lesions were found in the large intestine of the sheep. These data demonstrate the importance of using characteristic insoluble fallout simulants at levels to cause some deaths instead of depending on dosimeter measurements. An animal suffering from GI radiation injury will react quite differently physiologically from an animal under little or no radiation stress. Anorexia was accompanied by rumen stasis, which prevented the normal passage of ingesta. This was followed by severe diarrhea and weight loss.

Fallout irradiation injury to skin was observed[13] in cattle exposed at Alamogordo in 1945 and in cattle at the Nevada Test Site (D. S. Barth, personal communication, 1970). Minor-to-severe beta-irradiation injuries occurred although no lethalities were observed within 150 days in any of these cattle. Skin-irradiation injury appears similar to thermal burns except that the visible effects of thermal burns are immediate and the obvious effects of beta skin irradiation may not be observed for 3 or 4 weeks.

The skin-irradiation damage to the Alamogordo cattle was described by Brown, Reynolds, and Johnson[13] as the development of areas or zones of hyperkeratosis which formed plaques and cutaneous horns on the skin of the dorsa of the cattle. After 15 years three of the exposed cows developed squamous cell carcinoma of the skin in irradiation-damaged areas. In areas less severely affected, there was some alopecia and graying of the red hair. The location of these cattle in relation to the bomb is not known, but it is estimated that the radiation dose was 150 R gamma and 37,000 rads beta to the dorsal skin. There was no evidence of radiation damage on the ventral surfaces as has been predicted to result from the beta-bath exposure from radioactive fallout on the ground.

Combined Radiation Effects

Research on the effects of combining beta with gamma irradiation has recently been initiated with sheep (31 kg) and cattle (184 kg) at the UT–AEC Agricultural Research Laboratory. Results summarized in Table 1 show that these animals were much more susceptible to the combined radiation sources than to any one alone. Radiation levels chosen were slightly less than those expected to cause death from the ingested fallout simulant or the whole-body

Table 1

SHEEP AND CATTLE 60-DAY MORTALITY AFTER EXPOSURE TO SIMULATED FALLOUT

Treatment	Number of deaths*	
	Sheep	Cattle
Control	0	0
Whole body (WB)		
240 R ^{60}Co gamma at 1 R/min	0	0
Skin		
57,000 rads beta	1†	0
Gastrointestinal (GI)‡	1	2
GI + Skin	0	2
WB + Skin	0	0
WB + GI	3	5
WB + GI + Skin	4	8

*Eight animals were exposed to each treatment.

†Accidental death.

‡Sheep were fed 2.4 mCi of ^{90}Y-labeled sand per kilogram of body weight, and cattle were fed 2.0 mCi of ^{90}Y-labeled sand per kilogram of body weight.

gamma radiation. Using the NAS–NRC procedure,[8] we calculated the ingested level of 2.4 mCi per kilogram of body weight to simulate a 7% forage retention for sheep and that of 2 mCi/kg to simulate 9% forage retention for cattle exposed to 240-R gamma radiation. Whole-body gamma from six ^{60}Co sources[14] was used to give a bilateral air dose of 240 R at 1 R/min. Skin irradiation of the dorsa of these animals from flexible, sealed, beta-irradiation sources[15] gave a dose of approximately 57,000 rads to the surface of the hair or wool. The 7 to 9% forage retention levels are well within the range of 5 to 23% retention of 88- to 175-μ particles on alfalfa and pasture grasses.[16] Exposure of 12% of the body surface of sheep and 8% of the body surface of cattle provided a beta-to-gamma ratio comparable to the ratios estimated for the cattle exposed in 1945 at Alamogordo.[13] Skin irradiation alone under these conditions did not affect feed intake, but after 60 days skin-irradiated sheep weighed only 80% as much as the controls and as those exposed only to whole-body gamma irradiation. Sheep surviving a combination of whole-body gamma and skin and GI beta weighed only 60% as much as the controls in 60 days. Whole-body gamma radiation of 240 R at 1 R/min affected neither body weight nor feed consumption of sheep and cattle when no other radiation was given.

The importance of considering combined irradiation effects on survival of grazing livestock is convincing for the simulated-fallout conditions used to obtain the data summarized in Table 1. However, grazing livestock might be exposed to many different fallout conditions that would alter both mortality and productivity. For damage-assessment calculations, additional data are needed for alternative models, such as using different-size fallout-simulant particles, lower beta-energy exposures, different forage-retention levels, effects of absorbed isotopes (principally iodine), and different levels and rates of gamma exposures.

No data were found on the combined effects of beta and gamma irradiation in horses, swine, or poultry. Grazing equine might be severely affected by ingested fallout, but the damage would probably be greatest in the stomach and cecum. Alexander[17] described the gastric and cecal contractions in horses which would probably cause some stratification of ingesta, with the heavier fallout particles collecting in pockets as observed in the rumen and abomasum of cattle and sheep. Swine are normally fed in drylot and probably would not ingest enough radioactivity to increase losses that would occur above those from gamma irradiation alone. Data are not available on pasture-fed swine, but the effect would probably be minor. Ingested fallout would not be expected to be a problem in poultry production.

Data on lethality are meager for food-producing animals under simulated-fallout conditions. Estimates listed in Table 2 were obtained from published data on gamma lethality for the various species. Estimates for combined effects on cattle and sheep were made from research in progress at UT—AEC. Estimates for combined effects on swine, horses, and poultry were made by considering the grazing habits, anatomy, and physiology of these species since no data are available.

Table 2

ESTIMATED LIVESTOCK LETHALITY ($LD_{50/60}$) FROM FALLOUT-GAMMA-RADIATION EXPOSURE ALONE AND IN COMBINATION WITH BETA RADIATION*

	Total gamma exposure, R		
Animal	**Barn (WB)**	**Pen or corral (WB + Skin)**	**Pasture† (WB + Skin + GI)**
Cattle	500	450	180
Sheep	400	350	240
Swine	640	600‡	550‡
Equine	670	600‡	350‡
Poultry	900	850‡	800‡

*Data from M. C. Bell, L. B. Sasser, and J. L. West, Simulated-Fallout-Radiation Effects on Livestock, this volume.

†Assumed forage retention of 7 to 9%.

‡No data available; estimates are based on grazing habits, anatomy, and physiology of species.

RADIATION EFFECTS ON LIVESTOCK PRODUCTIVITY

Meat and Milk Production

Gamma radiation at levels below the lethal dose and at rates expected from fallout radiation would have minor to no measurable effects on livestock productivity. Animals surviving gamma radiation at dose rates of 0.1 to 2 R/min gain just as well as the controls,[7,18] and irradiated dairy cows produce almost as much milk as controls.[19,20] Lactation can, however, be reduced by ^{131}I destruction of thyroid tissue, as shown by Miller and Swanson,[21] who gave one of each pair of identical-twin dairy heifers doses of 99 to 180 μCi of ^{131}I per kilogram of body weight. These heifers averaged 305 kg at the time of treatment, and in their first lactation they averaged 54% of the production of the untreated twins. Radioisotopes of iodine are the major absorbed fission products of concern in early fallout during the first few weeks after detonation. However, grazing livestock are more likely to consume ^{131}I over a period of several weeks than in a single ingestion as described. Garner[22] estimated from data on sheep that cattle consuming 1500 μCi of ^{131}I daily would show a decline in milk yield and reduced viability of offspring. More-recent data indicate that cattle are less sensitive to ^{131}I injury than sheep.

Radioisotopes of iodine represent 15% of the total radioactivity 24 hr after fission, but most of these are short-lived isotopes.[23] Actual ^{131}I contributes only 0.8% at H + 24 hr and 3.5% at H + 4 days. Thyroid uptake by dairy cows at 24 hr reaches about 70% of the maximum uptake, which occurs at 72 hr.[24] Thus the decay factors and the rate of thyroid uptake would reduce the ^{131}I equivalent effective values to about 4% for H + 24 hr. The effectiveness of the radioiodine would be further reduced by the low solubility of early fallout. Comar, Wentworth, and Lengemann,[25] using a double tracer technique in six cows, found only 20% as much radioiodine from a fallout simulant in milk as from a soluble radioiodine. Ekman, Funkqvist, and Greitz[26] report 10% solubility for early fallout.

Neutron irradiation of 500 to 750 rads severely reduced feed intake, body weight, and milk production of dairy cows. Those exposed to the higher levels died within 40 days.[19] Nonlethal neutron irradiation of 300 rads significantly reduced growth of swine with no effect on feed intake.[27] However, neutron irradiation is not expected to be of significance compared with fallout radiation.

Fallout-simulant beta irradiation of the GI tract of cattle and sheep severely reduces feed intake and weight. Animals surviving this type of radiation usually return to normal feed consumption within 60 days, but considerably more time is required to recover the weight loss. In a UT–AEC study involving 32 sheep (31.1 ± 0.6 kg) fed a fallout simulant, five of the survivors developed abomasal hernias, and one developed a rumen fistula in the areas most severely affected by the beta radiation. These lesions did not develop until 60 days after treatment when the animals had regained appetites. Some of the lesions ruptured to the outside as late as 300 days after treatment. Rumen and abomasal tissue around these openings was firmly attached to the body cavity with no evidence of peritonitis and/or bacterial invasion. There is no evidence that these animals could not be used for food, especially if food were scarce. Preliminary results from experiments with cattle indicate that hernias and fistulae would not be a problem, because of the greater thickness of the tissue involved.

At present the research in progress with 184-kg beef calves indicates that feeding 2 mCi of ^{90}Y-labeled sand per kilogram of body weight for 3 days severely affects feed intake and body weight, but no calves died from either 240-R gamma at 1 R/min or from beta irradiation of 8% of the body surface over the dorsum. When these three treatments were combined, however, all calves died within 60 days (Table 1).

Neither beta irradiation to the skin nor whole-body gamma irradiation had an effect on feed intake, but weight gain was considerably reduced by skin irradiation of both sheep and cattle. During the winter months the loss of body heat would be expected to be much greater in a colder climate than in Tennessee, where the experimental animals had access to shelter. During the warm months the fly problem required frequent attention, starting about 30 days after skin irradiation. The fly-larvae damage could have caused increased animal losses if insecticides had not been used.

It appears that most surviving sheep and cattle suffering from skin injury from fallout or from GI injury in combination with whole-body gamma irradiation could eventually be used for food under emergency conditions. Research in progress at UT–AEC (Griffin and Eisele, personal communication, 1970) indicates that bacterial invasion is not a problem in swine dying from gamma irradiation given at a dose rate of 1 R/min. Until more data are available, it is recommended that, for 15 to 60 days after exposure to levels to cause some mortality, only muscle meat from surviving animals be used for food.

Poultry

A review by Wetherbee[28] showed that young irradiated chicks developed hypotension and that the survivors had a reduced rate of growth. Egg production was reduced only when layers were exposed to 600 R and above from ^{60}Co at 0.9 R/min, and the survivors gradually regained their normal levels of egg production. Most of the reduction in egg production occurred between days 11 and 20. More recently Maloney and Mraz[29] showed that survivors of a group of White Leghorn hens exposed to 400 to 800 R ^{60}Co at 5 R/min had a 10-day temporary drop in egg production starting 10 days after exposure. This drop in egg production lasted 40 days when the total dose remained constant and the dose rate was increased to 45 R/min.

Exposure of incubated, fertilized eggs to less than 80 R of X rays accelerated the development of the embryo, but higher doses retarded development. Hatchability increases of 10% over the controls have been claimed by using exposures of up to 30 R of X rays.[28] The unincubated, fertile egg is relatively resistant to gamma-radiation effects. Sensitivity increased the first 3 days of development then decreased through day 12, when it leveled off at an $LD_{50/30}$ of about 750 R. A second period of radiation sensitivity was found at incubation day 18.

Beta irradiation of the GI tract and skin of poultry would not be expected to be a problem in poultry production. Even the few turkeys on the range depend mostly on feed supplements and very little on range pasture.

Reproduction

Studies of radiation effects on reproduction in food-producing animals have rightly been concentrated on gamma radiation. Neither ingestion nor skin irradiation from beta particles would be expected to have a direct effect on reproduction, but there could be abscopal effects in addition to anorexia and weight loss.

Whole-body ^{60}Co gamma radiation of beef heifers has not affected the long-term reproductive performance of 179 survivors for 8 years postirradiation.[18] Acute radiation sickness and mortality occurred in a large number of these cattle exposed to 200 to 400 R at 0.7 R/min from ^{60}Co; however, there were no differences that could be attributed to radiation in the performance of offspring in comparison with offspring of the 40 controls. Neither did exposure of beef cows to the first atomic bomb at Alamogordo have a measurable effect on reproductive performance. The ovaries, which are well protected in adult cows, would receive about 40% of the air dose. In addition, it has been estimated that over twice the lethal level given directly to the ovaries would be required to sterilize females.[30,31] There is a high percentage of bone deformities in offspring of pregnant females gamma irradiated with 100 rads or more during a short period in their gestation: gestation days 32 to 34 in cattle and 22 to 24 in sheep. At this stage of development, the limb buds are just starting to form in the embryos, and they are very sensitive to gamma radiation.[18,32,33]

The developing fetus concentrates ^{131}I much more than its dam,[34] and fetal thyroid takes up almost as much ^{131}I as the dam thyroid.[35] However, thyroid insufficiency can be counteracted by using thyroxin or by feeding iodinated casein if the thyroid is damaged by ^{131}I irradiation.

Males surviving fallout gamma radiation at levels of 200 R or more would be expected to be temporarily sterile starting about 6 weeks after exposure, but this would last for only a few weeks.[36] Since a large number of females can be bred to one male either naturally or through artificial insemination, male sterility is not expected to be a problem in food-producing animals.

Work

Shetland ponies surviving gamma-radiation exposure of 50 R/week at 25 R/hr for a total of 650 R have been used in the study of radiation effects on work performance and several other physiological parameters. After recovery from the early radiation effects, the irradiated ponies performed as well as their control teammates over a period of 2 years.[18]

Genetic and Life-Span Effects

Although gamma-irradiated animals show an increase in chromosome aberrations,[37] the chance of observing genetic changes in offspring of large animals is rather small. Mullaney and Cox[38] reported that pigs sired by boars after they had recovered from 300 R of X rays to the testes were not adversely affected. In these studies involving over 3000 litters of pigs, irradiation of the maternal grandsire decreased ($P < 0.01$) the number of stillborn pigs in one of the two breeds studied. Survivors of the lifetime Hereford cows at UT–AEC (discussed in the section on reproduction) show no indication of genetic effects on offspring over the past 8 years of observation.[18]

Since unproductive food-producing–breeding animals are normally culled and used for food, there is little concern for life-span effects in large animals unless there is a shortage of breeding animals. From the limited data available,[18] it appears that life-span and productive life-span of swine and cattle are slightly reduced in long-term survivors of whole-body radiation. Life-span lengthening of 37% in males and 16% in females has been reported in 10 generations of mice irradiated from drinking water containing 1 μCi of ^{90}Sr and 4 μCi of ^{137}Cs per liter. These mice also showed improved reproductive efficiency in the study using 255 litters. Mice drinking water with 100 times these concentrations of ^{90}Sr and ^{137}Cs showed adverse effects on both life-span and reproduction.[39] No data were found on large animals subjected to these types of tests.

COUNTERMEASURES

The countermeasures that can be recommended to save the largest number of grazing food-producing animals in a heavy fallout field are sheltering and using stored feed. In an area where gamma irradiation alone would be lethal to a small percentage of grazing animals, any shelter that groups and restricts the animals provides mutual shielding,[40] prevents them from grazing pastures contaminated with early fallout, and probably ensures that most cattle and sheep would survive instead of dying from exposure to a combination of whole-body gamma and beta irradiation to the skin and GI tract.

Shelters providing large protection factors would be desirable but are not available on most farms, as shown in a pilot survey in Tennessee.[41] Buildings available for shelters on these farms gave an average protection factor of only 1.8, but the real importance of these buildings would be to prevent fallout damage to the skin, to prevent ingestion of forage contaminated with high levels of fallout, and to provide mutual shielding.

The limited data available show that preventing livestock from eating contaminated feed during the first 72 hr after fallout arrival is one of the most important countermeasures available. Anorexia, diarrhea, and GI injury and perhaps thyroid injury could greatly increase the lethality percentage. Weight and productivity of the survivors of grazing livestock would be severely affected. If no shelter were available, confining animals in a fenced corral, ravine, or woods would be desirable to increase mutual shielding and prevent ingestion of forage contaminated with early fallout.

Skin damage from fallout increases heat loss and parasitic problems but is probably of less consequence than beta ingestion and whole-body gamma damage. The USSR textbook[10] recommends blankets and canvas as improvised means of protecting the skin of animals. It was also suggested that valuable breeding animals could get added protection from a chemically treated, protective muzzle bag that would prevent the animal from eating contaminated feed and reduce the radioactivity inhaled when animals are being taken out of a contaminated area.

ACKNOWLEDGMENTS

The UT–AEC Agricultural Research Laboratory is operated by the Tennessee Agricultural Experiment Station for the U. S. Atomic Energy Commission under Contract AT-40-GEN-242.

This work was supported in part by the U. S. Office of Civil Defense and is published with the permission of the Dean of the University of Tennessee Agricultural Experiment Station, Knoxville.

REFERENCES

1. N. P. Page, The Effect of Dose Protraction on Radiation Lethality of Large Animals, in Proceedings of a Symposium on Dose Rate in Mammalian Radiation Biology, Apr. 29–May 1, 1968, Oak Ridge, Tenn., D. G. Brown, R. G. Cragle, and T. R. Noonan (Eds.), USAEC Report CONF-680410, pp. 12.1–12.23, UT–AEC Agricultural Research Laboratory, July 12, 1968.
2. E. J. Ainsworth, N. P. Page, J. F. Taylor, G. F. Leong, and E. T. Still, Dose Rate Studies with Sheep and Swine, in Proceedings of a Symposium on Dose Rate in Mammalian Radiation Biology, Apr. 29–May 1, 1968, Oak Ridge, Tenn., D. G. Brown, R. G. Cragle, and T. R. Noonan (Eds.), USAEC Report CONF-680410, pp. 4.1–4.21, UT–AEC Agricultural Research Laboratory, July 12, 1968.
3. D. G. Brown and R. G. Cragle, Some Observations of Dose-Rate Effect of Radiation on Burros, Swine, and Cattle, in Proceedings of a Symposium on Dose Rate in Mammalian Radiation Biology, Apr. 29–May 1, 1968, Oak Ridge, Tenn., D. G. Brown, R. G. Cragle, and T. R. Noonan (Eds.), USAEC Report CONF-680410, pp. 5.1–5.16, UT–AEC Agricultural Research Laboratory, July 12, 1968.
4. M. T. Case and J. Simon, Whole-Body Gamma Irradiation of New-Born Pigs: The $LD_{50/30}$, *Amer. J. Vet. Res.*, **31:** 113–115 (1970).
5. D. G. Brown, Clinical Observations on Cattle Exposed to Lethal Doses of Ionizing Radiation, *J. Amer. Vet. Med. Ass.*, **140:** 1051–1055 (1962).

6. F. W. Chambers, C. R. Biles, L. J. Bodenlos, and J. H. Dowling, Mortality and Clinical Signs in Swine Exposed to Total-Body Cobalt-60 Gamma Irradiation, *Radiat. Res.,* **22:** 316–333 (1964).
7. R. S. Lowrey and M. C. Bell, Whole-Body Irradiation in the Young Pig: Growth, Hematology and Metabolism of ^{45}Ca and ^{89}Sr, *Radiat. Res.,* **23:** 580–593 (1964).
8. National Academy of Sciences–National Research Council, Damage to Livestock from Radioactive Fallout in Event of Nuclear War, Publication 1078, Washington, D. C., 1963.
9. M. M. Nold, R. L. Hayes, and C. L. Comar, Internal Radiation Dose Measurements in Live Experimental Animals, *Health Phys.,* **4:** 86–100 (1960).
10. *Civil Defense in Rural Regions,* Military Publishing House of USSR Ministry of Defense, Moscow, 1965.
11. M. C. Bell Airborne Radionuclides and Animals, in *Agriculture and the Quality of Our Environment,* N. C. Brady (Ed.), American Association for the Advancement of Science, Symposium No. 85, pp. 77–90, Washington, D. C., 1967.
12. M. C. Bell, L. B. Sasser, J. L. West, and L. Wade, Jr., Effects of Feeding ^{90}Y-Labeled Fallout Simulant to Sheep, *Radiat. Res.,* **43:** 71-82 (1970).
13. D. G. Brown, R. A. Reynolds, and D. F. Johnson, Late Effects in Cattle Exposed to Radioactive Fallout, *Amer. J. Vet. Res.,* **27:** 1509–1514 (1966).
14. J. S. Cheka, E. M. Robinson, L. Wade, Jr., and W. A. Gramly, The UT–AEC Agricultural Research Laboratory Variable Gamma Dose-Rate Facility, *Health Phys.,* **20:** 337-340 (1971).
15. M. C. Bell, Flexible Sealed ^{90}Sr–^{90}Y Sources for Large Area Skin Irradiation, *Int. J. Appl. Radiat. Isotop.,* **21:** 42–43 (1970).
16. J. E. Johnson and A. I. Lovaas, Deposition and Retention of Simulated Near-In Fallout by Food Crops and Livestock, First Technical Progress Report, Colorado State University, Fort Collins, Colo., 1969.
17. F. Alexander, Digestion in the Horse, in *Progress in Nutrition and Allied Sciences,* D. P. Cuthbertson (Ed.), pp. 259–268, Oliver and Boyd, Ltd., Edinburgh, 1963.
18. UT–AEC Agricultural Research Laboratory, Annual Progress Report, Jan. 1–Dec. 31, 1968, USAEC Report ORO-672, October 1969.
19. R. G. Cragle, J. K. Miller, E. W. Swanson, and D. G. Brown, Lactation and Radionuclide Metabolism Responses of Dairy Cattle to Lethal Doses of Gamma and Neutron Radiation, *J. Dairy Sci.,* **48:** 942–946 (1965).
20. E. W. Swanson, R. G. Cragle, and J. K. Miller, Effects of Irradiation upon Lactation, *J. Dairy Sci.,* **48:** 563–568 (1965).
21. J. K. Miller and E. W. Swanson, Effects of Iodine-131 Thyroid Damage on Lactation and Thyroid Function in the Bovine, *J. Dairy Sci.,* **52:** 95–100 (1969).
22. R. J. Garner, Environmental Contamination and Grazing Animals, *Health Phys.,* **9:** 597–605 (1963).
23. R. V. Petrov, V. N. Pravetskii, Yu. S. Stepanov, and M. I. Shal'nov, Radioactive Fallout. Physics, Biological Effects, and Protective Measures, USAEC Report AEC-tr-6634, translation of Zashchita ot Radioaktivnykh Osadkov, Gosudarstvennoe Izdatel stvo, Meditsinskoi Literatury, Moscow, 1963.
24. E. W. Swanson, F. W. Lengemann, and R. A. Monroe, Factors Affecting the Thyroid Uptake of ^{131}I in Dairy Cows, *J. Anim. Sci.,* **16:** 318–327 (1957).
25. C. L. Comar R. A. Wentworth, and F. W. Lengemann, A Study of Metabolism of Selected Radionuclides Fed in Various Physical Forms to Dairy Cows, Final Report, OCD Report TRC-67-33, Cornell University, Ithaca, N. Y., 1967.
26. L. Ekman, B. Funkqvist, and U. Greitz, Beta- and Gamma-Dose Measurements in the Gastrointestinal Tract of Goats with LiF Dosimeters After a Single Intake of Simulant Mixed Fission Products, Swedish Report FOA-4-4418, March 1970.

27. H. Rosas and M. C. Bell, Effects on Swine of Unilateral Neutron Irradiation, Calcium and Zinc, *Radiat. Res.*, **39**: 164–176 (1969).
28. D. K. Wetherbee, Gamma Irradiation of Birds' Eggs and the Radiosensitivity of Birds, USAEC Report TID-24521, Massachusetts Agricultural Experiment Station, October 1966.
29. M. A. Maloney and F. R. Mraz, The Effect of Whole-Body Gamma Irradiation on Survivors' Egg Production in the White Leghorn and Bobwhite Quail, *Poultry Sci.*, **48**: 1939-1944 (1969).
30. B. H. Erickson, Radioresponse of the Prepuberal Porcine Ovary, *Int. J. Radiat. Biol.*, **13**: 57–67 (1967).
31. B. H. Erickson, Effect of Gamma Radiation on the Prepuberal Bovine Ovary, *Radiat. Res.*, **31**: 441–451 (1967).
32. B. H. Erickson and R. L. Murphree, Limb Development in Prenatally Irradiated Cattle, Sheep, and Swine, *J. Anim. Sci.*, **23**: 1066–1071 (1964).
33. A. F. McFee, R. L. Murphree, and R. L. Reynolds, Skeletal Defects in Prenatally Irradiated Sheep, Cattle and Swine, *J. Anim. Sci.*, **24**: 1131–1135 (1965).
34. P. W. Aschbacher, R. G. Cragle, E. W. Swanson, and J. K. Miller, Metabolism of Oral Iodine and 3,5-diiodosalicylic Acid in the Pregnant Cow, *J. Dairy Sci.*, **49**: 1042–1045 (1966).
35. J. K. Miller, E. W. Swanson, P. W. Aschbacher, and R. G. Cragle, Iodine Transfer and Concentration in the Prepartum Cow, Fetus and Neonatal Calf, *J. Dairy Sci.*, **50**: 1301–1305 (1967).
36. B. H. Erickson, Radiation Effects on Gonadal Development in Farm Animals, *J. Anim. Sci.*, **24**: 568–583 (1965).
37. A. F. McFee, M. W. Banner, and Mary N. Sherrill, Influence of Animal Age on Radiation-Induced Chromosome Aberrations in Swine Leukocytes, *Radiat. Res.*, **41**: 425–435 (1970).
38. P. D. Mullaney and D. F. Cox, Effects of Paternal X-Irradiation on Litter Size and Early Postnatal Mortality in Swine, *Mutat. Res.*, **9**: 337–340 (1970).
39. K. Nishio, T. Megumi, and M. Yonezawa, Effects of ^{137}Cs and ^{90}Sr Administered Continuously and Through Generations upon Mice. VI. *Ann. Rep. Radiat. Center Osaka Prefect.*, **9**: 86–93 (1968).
40. K. Mills and K. Evans, Protecting Livestock from Radioactive Fallout, University of Kentucky Extension Service Miscellaneous Publication 338, 1966.
41. S. A. Griffin, in Civil Defense Research Project. Annual Progress Report March 1968–March 1969, USAEC Report ORNL-4413(Pt. 1), pp.94–96, Oak Ridge National Laboratory, October 1969.

THE EFFECTS OF EXTERNAL GAMMA RADIATION FROM RADIOACTIVE FALLOUT ON PLANTS, WITH SPECIAL REFERENCE TO CROP PRODUCTION

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ABSTRACT

This paper describes the major problems involved in attempting to predict for economically useful plants the degree of radiation damage that would arise from exposure to high-level radioactive fallout. Since almost no data exist on the deleterious effects inflicted on crops by actual fallout radiation, it is necessary to extrapolate from the existing radiobotanical data concerned with the effects of gamma radiation on survival and yield of plants.

A number of factors can modify the effects of the radiation and hence influence the accuracy of predictions of postattack injury. The most important variables are (1) species differences in interphase chromosome volume (the larger this value, the more sensitive the plant), (2) exposure rate (high rates are more effective than lower rates), (3) stage of development of the plant (a complex and difficult variable to assess), (4) postirradiation time (generally the longer the time, the greater the degree of damage), and (5) numerous environmental factors such as moisture, temperature, light, competition, etc., which normally modify plant growth and yield. These factors, acting singly or in various combinations, can have a considerable effect on the radiation response and thereby make more difficult the prediction of postattack injury.

Survival and yield data obtained from irradiation of growing plants are presented for many species. The most useful values in comparing sensitivities are LD_{10}, LD_{50}, and LD_{90} (exposures required to reduce survival by 10, 50, and 90%), and YD_{10}, YD_{50}, and YD_{90} (exposures required to reduce yield by 10, 50, and 90%). A log-log regression of LD_{10} vs. YD_{50} for 36-hr fallout-decay-simulation (FDS) gamma exposures has a slope not significantly different from +1; this indicates that, in general, an exposure producing an LD_{10} will reduce yield by 50%. Other LD_{10} values may also be predicted from regressions of interphase chromosome volume on LD_{10}.

Predicted YD_{50} values following FDS exposures are given for 89 crop plants and for 82 woody plants for a 16-hr constant-rate exposure. Using these predictions and the available radiobiological data, we can draw some conclusions concerning the vulnerability of crop plants to fallout radiation. The cereals (wheat, barley, oats, and maize), which are probably our most important group of crop plants, would be the most sensitive, having YD_{50} values ranging from about 1 to 4 kR (rice is much more resistant). The legumes (peas and beans) include both sensitive and resistant species, having YD_{50} values ranging from less than 1 to 12 kR. Root crops (onions, garlic, beets, potatoes, and radishes) have a wider range in sensitivity; YD_{50} values range from 1 to 16 kR. For pasture and forage crops, YD_{50} varies from 2 to 20 kR. Of the herbaceous crop species, 70% fall in the predicted sensitivity range between 4 and 16 kR. Woody species have a range of predicted LD_{50} values between about 0.4 and 8 kR, the gymnosperms predominating below 2 kR.

These predictions are for average conditions only. We still lack a significant amount of radiobiological data required to make confident predictions of the expected response of many species to high-level fallout-gamma exposure. Also, inadequate information about beta-radiation injury and its possible interaction with gamma radiation makes extrapolation to actual fallout conditions even more difficult.

Plants in areas receiving radioactive fallout will be exposed to two types of external radiation, gamma and beta; the relative biological effectiveness of these two types of radiation was recently shown[1] to be approximately 1. In areas of heavy fallout, either type of radiation alone could seriously reduce the growth or yield of plants, at least at certain stages of plant development. Under conditions of lighter fallout, the combined exposures from both types of radiation could also produce very serious effects, up to complete destruction of some crops. However, this report reviews only known or expected effects of gamma radiation on various species of plants given a range of exposures at one or more stages in their life cycles. The hazards of direct contamination of foodstuffs by fallout radionuclides have been discussed elsewhere.[2–5] The long-lived nuclides are not now considered as serious a hazard as was previously thought.[6] Although the dislocations in agricultural practices and food distribution associated with other disturbances and/or the reduced availability of manpower and horsepower which would result from a nuclear war are important in the overall context of postattack recovery, we shall not consider them here.

Previous studies on the effects of gamma radiation on growing plants are many and varied.[7–14] Unfortunately, however, many different exposure rates have been given under differing conditions with various criteria of effect being used. No previous attempt has been made to assemble the majority of the pertinent data and devise a means of presenting them in a uniformly comprehensible manner. This paper reviews the major modifying factors, such as exposure rate and duration, stage at irradiation, environmental conditions, etc.; surveys the available pertinent data; indicates what currently appear to be the general trends of response to fallout or simulated-fallout gamma radiation; and predicts the probable responses for plant species for which no data are currently available.

BACKGROUND INFORMATION

The wide range of radiosensitivity among different plant species to external X or gamma radiation is well documented.[11,12,14–20] Radiosensitivity varies by at least 100-fold among species and by over 50-fold within a species irradiated at different stages. Certain stages of flower-bud development are known to be much more sensitive than others and also more sensitive than meristem cells in the vegetative stage.[20–22] Radiation injury expresses itself after a few days, weeks, or, in some cases, years as abnormal shape or appearance, reduced growth or yield, loss of reproductive capacity, sometimes wilting, and, finally, at the higher exposures death. Although we recognize the importance of genetic effects, we shall not attempt here to survey the vast body of literature on this subject.

It is now known that the wide range in sensitivity of plant species irradiated and grown under uniform experimental conditions can be attributed largely to variation in the size of the chromosomes of the plants.[11,16,19,23–29] A direct relation between chromosome size (measured as the average volume of an interphase chromosome) and sensitivity to gamma radiation given under specified conditions has been established, showing that, as the size of the chromosomes increases from one species to another, the amount of radiation required to produce a specified effect decreases (Figs. 1 and 2). The consistency of this relation is the basic premise on which our predictions are based [see Radiosensitivity Predictions (Based on ICV Data)].

When plants are irradiated under uniform conditions with a range of exposures which, depending on their magnitude, will produce measurable decreases in yield and/or survival, response curves can be obtained. Values not actually observed to occur at any of the exposures given can be calculated from these data. Survival end points that have been found to be most useful in describing radiation effects on plants are LD_{10}, LD_{50}, LD_{90}, and LD_{100}; the exposures required to reduce plant survival by 10, 50, 90, and 100%, respectively. Similarly, for yield reduction the particular end points of most use are YD_{10}, YD_{50}, and YD_{90}, the exposures required to reduce growth or yield by 10, 50, and 90%, respectively.

There is extensive literature on growth stimulation in plants after exposure to ionizing radiation, and some investigators claim statistically significant increases in yield after exposures usually referred to as low doses, although a wide range of exposures is used. Surveys of the available data have been given in various publications.[7,30–33] In our opinion the probability of beneficial effects of fallout radiation on crop yield is so far outweighed by the probability of deleterious effects that no further consideration will be given here to possible enhanced growth or yield.

A particularly useful relation between a survival end point and a yield end point is shown in Fig. 3. For all practical purposes, for the plant species studied, the exposure that produces an LD_{10} also produces a YD_{50}. Thus the determination of the LD_{10} for any species should provide a fair approximation of the YD_{50}. This is an advantage because determination of YD_{50} generally requires experiments of greater magnitude, with better facilities and more manpower. Also, survival data for a crop for which no yield data exist can be converted to an estimated effect on yield.

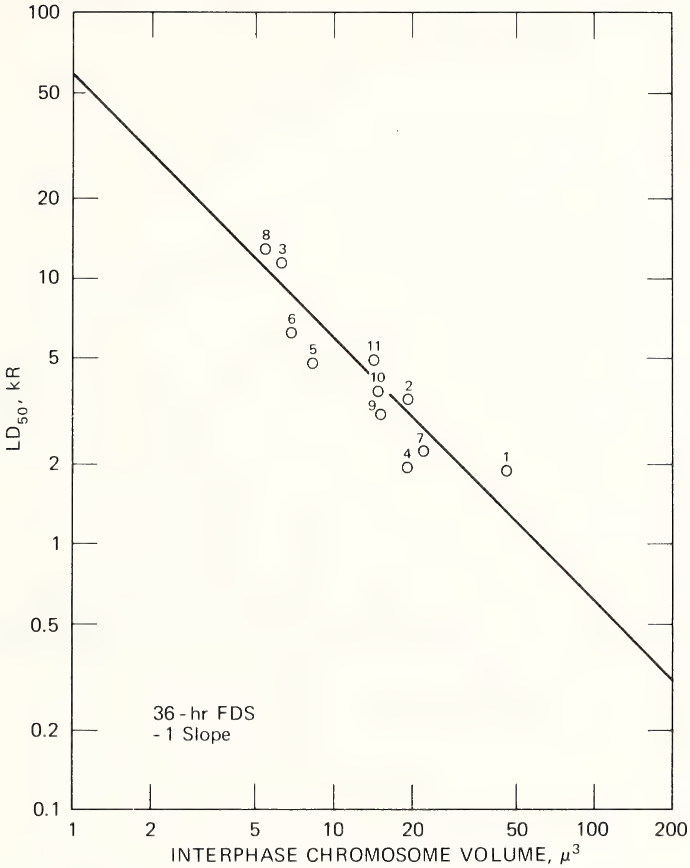

Fig. 1 Log-log regression of LD_{50} against ICV for 10 species of economic plants given a 36-hr FDS exposure as young seedlings.

1 *Allium cepa*
2 *Avena sativa*
3 *Brassica oleracea*
4 *Hordeum vulgare*
5 *Lactuca sativa*
6 *Phaseolus limensis*
7 *Pisum sativum*
8 *Raphanus sativus*
9 *Triticum aestivum*
10 *Zea mays*
11 *Zea mays*

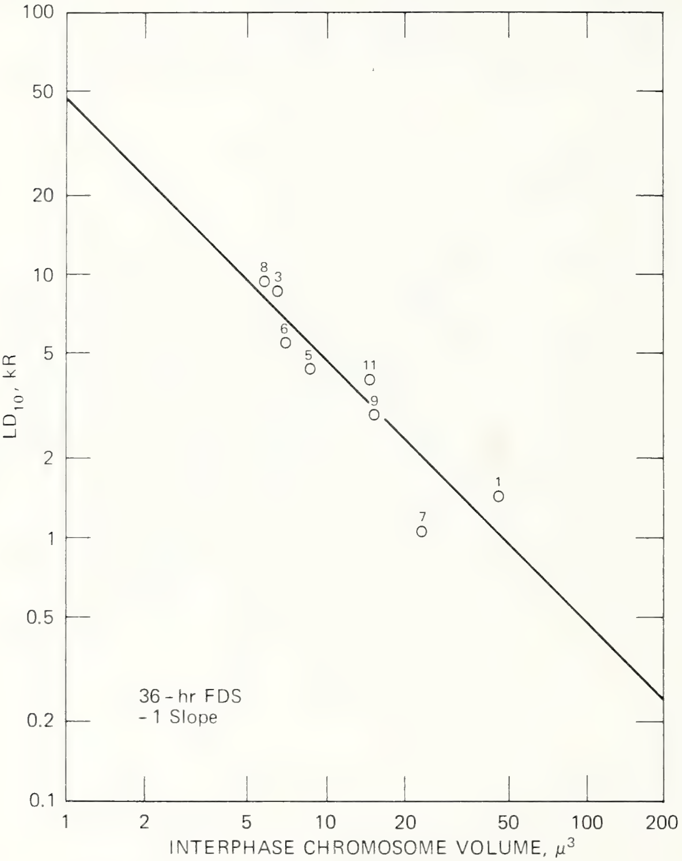

Fig. 2 Log-log regression of LD$_{10}$ against ICV for eight species of economic plants given a 36-hr FDS exposure as young seedlings.

1	*Allium cepa*	7	*Pisum sativum*
3	*Brassica oleracea*	8	*Raphanus sativus*
5	*Lactuca sativa*	9	*Triticum aestivum*
6	*Phaseolus limensis*	11	*Zea mays*

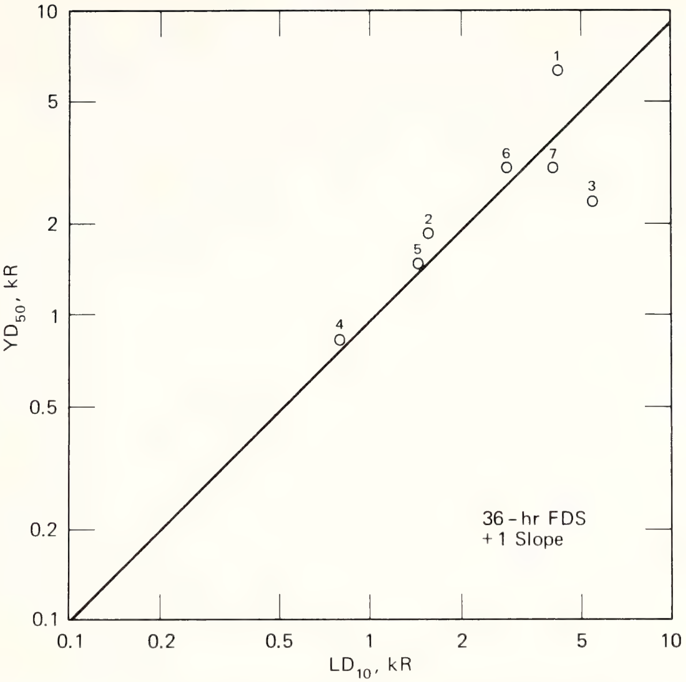

Fig. 3 Log-log regression of YD_{50} against LD_{10} for six species of economic plants given a 36-hr FDS exposure as young seedlings.

1	*Cucurbita pepo*	5	*Pisum sativum*
2	*Hordeum vulgare*	6	*Triticum aestivum*
3	*Phaseolus limensis*	7	*Zea mays*
4	*Pisum sativum*		

Other effects of importance for consumable economic crops are changes in starch,[34] sugar (personal communication from R. S. Russell and Ref. 35), and protein[36] content and minor variations such as differences in taste,[37-39] shape,[40-42] color,[43] and perhaps wholesomeness.[44]

MODIFYING FACTORS

General Considerations

Basic research in radiobiology has shown that there are many biological, radiological, and environmental factors that determine or modify radiosensitivity. A partial list of these factors is given in Table 1; no indication of the extent or direction of the change in sensitivity is given, however. To emphasize the possible significance of such factors, we have made estimates of the degree of

Table 1

BIOLOGICAL, RADIOLOGICAL, AND ENVIRONMENTAL FACTORS THAT CONTRIBUTE TO VARIATIONS IN RADIOBIOLOGICAL RESPONSES OF PLANTS*

Biological factors

- Cytological and genetic
  - Chromosome number
  - Chromosome volume
  - DNA content per chromosome
  - Heterochromatin (amount of)
  - Genotype or taxonomic group
  - Length of mitotic cycle
  - Percentage of cells dividing
  - Stage of nuclear cycle (especially in meiosis)
- Morphological organization and development
  - Type of cell or tissue
  - Stage of differentiation (e.g., vegetative or floral)
  - Portion(s) of plant irradiated
  - Size of plant or depth of sensitive organs
- Physiological or biochemical
  - Age of plant
  - Metabolic rate
  - Stage of growth cycle (active or dormant)
  - pH of cells (and soil)
  - Nutritional state
  - Concentration of growth hormones
  - Concentration of protective or sensitizing substances

Radiological factors

- Kinds of radiation(s)
- Energy or LET of radiation
- Exposure fractionation and previous exposures
- Exposure rate
- Exposure duration
- Depth dose
- Location of radioisotope
- Shielding (various)
- Relative humidity
- Moisture content of soil and plants
- Density of soil
- Chemical composition of plants and soil (for neutrons)
- Distance from detonation
- Time after detonation

Environmental factors

- Temperature
- Wind velocity
- Dust or fallout (amount and particle size)
- Moisture content of air, soil, and plants
- Insects or other pests
- Competition (other plants)
- Season (day length, etc.)
- Available sunlight
- Soil fertility

*Modified from Gunckel and Sparrow.[8]

modifying effect of a few of the more important ones that might apply in an actual fallout situation. These, along with the accumulated effect of all the factors acting in the same direction, are given in Table 2. Of course, the probability that all these factors would simultaneously act in the same direction is remote. However, the exercise clearly emphasizes why we cannot assign an absolute sensitivity value to a given crop or species unless most of the radiological, biological, and environmental conditions are clearly stated.

Table 2

MAJOR FACTORS THAT DETERMINE OR MODIFY RADIOSENSITIVITY OF PLANTS AND EXTENT OF EFFECT PRODUCED BY EACH FACTOR WHEN ALONE AND WHEN CUMULATED WITH ALL OTHER FACTORS (ASSUMING THEM TO BE CUMULATIVE)

Factor	Change that increases effect	Maximum (or estimated) effect	Maximum cumulative interaction
Species (chromosome size*)[16]	Larger ICV	100	100
Stage or age†	Various	50	5,000
Environmental	Various	5‡	25,000
Exposure rate[44]	Higher rates	4	100,000
$\beta + \gamma$ interaction	Combination	2‡	200,000
RBE[90]	More densely ionizing radiation	20	4,000,000

*ICV (interphase chromosome volume).

†See Table 4.

‡Estimates considered to be conservative.

Table 3

RATIOS OF LD_{50} VALUES* FOR VARIOUS EXPOSURE TIMES WITH CONSTANT-RATE, FDS, AND BU + FDS EXPOSURES (THE 16-HR LD_{50} BEING GIVEN AN ARBITRARY VALUE OF 1.00)

Exposure time, hr	Treatment: Constant rate	Treatment: Fallout decay simulation	Treatment: Buildup + FDS
36	0.76†	1.40	1.41
16	1.00		
8	1.40		
4	2.00		
1	2.70		

*Based on data from lettuce irradiations[37] except where noted.

†Based on data from squash, cabbage, pea, and maize irradiations.[39,51]

Influence of Exposure Rate and Duration

Exposure rate and duration are major variables which, under many conditions, modify the extent of injury produced by a given amount of radiation. Though studies done on the same species with different rates of exposure are desirable, they are not often made. However, as shown in Table 3, a given exposure delivered at a higher rate (shorter exposure time) is more effective than the same exposure at a lower rate (longer exposure time). There are some limits to this effect, however. At very high exposure rates, further increases in rate may not bring about additional increases in effect,[45–47] and at very low rates a point is reached where no external differences between irradiated and nonirradiated plants can be detected.[18,48,49] This complete range in exposure-rate effects has recently been reported in one system.[50] Unfortunately current knowledge of exposure-rate effects is generally too inadequate to allow the application of mathematical models that would permit the prediction of effects at several different exposure rates from the results obtained at one exposure rate since the critical exposure rate may vary from species to species. For these reasons, the actual conditions of exposure for each experiment reported have been given when available since they do differ considerably.

Recent data show that for equal total exposures a 36-hr fallout-decay-simulation (FDS) treatment with decreasing exposure rates is more effective in reducing survival and yield than the previously used standard 16-hr constant-rate (CR) treatment.[39,45,51] The average ratio of 16-hr CR to FDS treatment for several crop species at the LD_{50} exposure is 1.4 (Table 3). The greater effectiveness of the FDS treatment is due to the very high initial exposure rates encountered with this type of exposure.[45] For yield reduction the FDS is more effective only at the higher exposures. It has been shown also that there was no significant difference between equal total exposures of an FDS treatment and an 8-hr CR treatment.[45] This is attributable to the fact that there is very little difference between the average exposure rate for an FDS treatment and the exposure rate for an 8-hr CR treatment. With exposure times less than 8 hr, the effectiveness of a given exposure increases with decreasing time (Table 3; see also Tables 8, 9, and 12).

Influence of Age and Stage Irradiated

It is well known that the age of a plant or its stage of differentiation or development can have a major influence on the amount of radiation required to produce a common end point.[52–62] The significance of stage of development at the time of irradiation is clearly indicated in Table 4, which gives data for sensitivity of various stages of development of the corn plant. The data presented indicate that the difference between the most sensitive stage (meiosis) and the most resistant stage (dry seed) exceeds 50-fold. Fortunately in most plant species the highly sensitive stage of meiosis is a fairly short one, lasting at most a few days. The high radiosensitivity of pollen may be important, especially since all of the most important cereal crops are wind pollinated. Because of their small size, most pollen grains would be vulnerable to injury from beta radiation both on the plant and in the air. Since the beta dose might exceed the gamma dose in most fallout situations, the total effect on pollen

Table 4

RADIOSENSITIVITY OF VARIOUS DEVELOPMENTAL STAGES OF MAIZE (*ZEA MAYS*)

Stage	End point	Exposure, kR	Duration and type of exposure
Dry seed[91]			
10.6% moisture	LD_{50} (survival at 20 days)	54	2.7 kR/min gamma
1.9% moisture		10	2.7 kR/min gamma
Young plants	50% reduction in seed yield†	1	50 R/min gamma
	LD_{50} (at maturity)[51]	5.1	16 hr acute gamma
	LD_{100} (at maturity)[51]	6.5	16 hr acute gamma
	10% reduction in seed yield[51]	1	16 hr acute gamma
	50% reduction in seed yield[51]	4.3	16 hr acute gamma
	100% reduction in seed yield[51]	6	16 hr acute gamma
Meiosis	43% reduction in fresh weight of offspring†	1*	50 R/min gamma
Pollen (mature)	LD_{50} for flowers producing seed[92]	1.2	1.2 kR/min gamma

*Varies with stage of meiosis. Meiotic prophase is very sensitive but is of short duration.
†M. J. Constantin, UT–AEC Agricultural Research Laboratory, unpublished data, 1970.

could have a significant effect on yield, at least for the more sensitive species. This would also be true for very small seedlings or plant parts small enough for penetration by beta radiation.

The variation in sensitivity measured as reduced yield after irradiation at several stages during the growing period is given for five major crops in Fig. 4. The sensitivity for each crop can vary during the growing period from almost no effect to total loss of yield after identical exposures. Each crop has its own characteristic period of peak sensitivity, which varies from 6 days after emergence for soybeans to 195 days after emergence for winter barley. These data indicate not only the degree of variation in sensitivity with stage for a single species but also the variation among species. Not all these crops, however, would be expected to be at their stage of maximum sensitivity in a specific fallout situation. Differences in sensitivity with respect to yield of various economic plants irradiated at different stages of development are given in the section on deleterious effects on yield and survival of economic plants.

Influence of Postirradiation Time

Of considerable significance, particularly for economic plants, is the time after irradiation at which the radiation effects first become evident or first produce a serious effect. There are wide variations among species in the timing of specific responses to irradiation.[63] Some plants show adverse effects or die

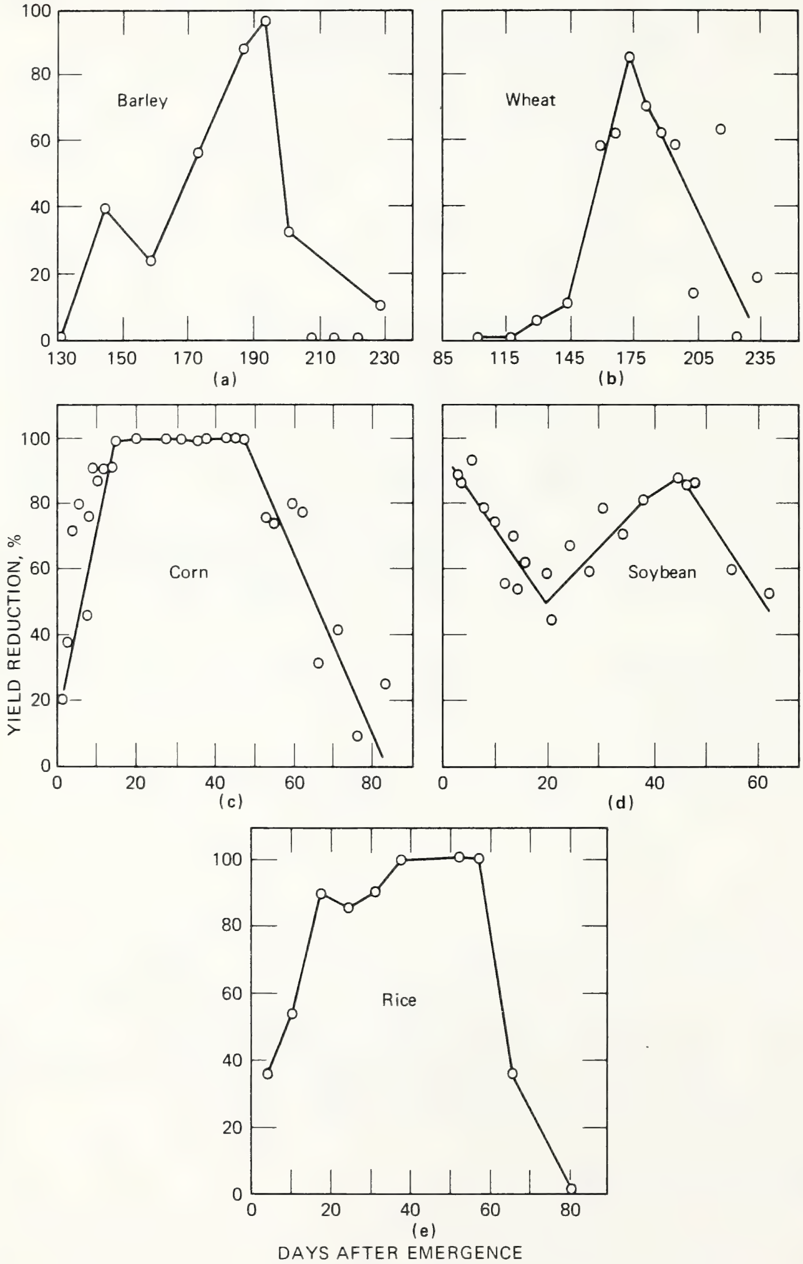
Barley
Wheat
Corn
Soybean
Rice
YIELD REDUCTION, %
0
20
40
60
80
100
130
150
170
190
210
230
(a)
85
115
145
175
205
235
(b)
0
20
40
60
80
(c)
0
20
40
60
(d)
0
20
40
60
80
(e)
DAYS AFTER EMERGENCE

Figure 4

within a few days or at most a few weeks after irradiation, whereas others do not manifest such effects for many months, or even years for woody plants.[11] Results of experiments with tomato have shown that fruit production is considerably delayed by irradiation and the extent of delay increases with increasing exposure (Fig. 5). When the growing season is short, such a delay in production or ripening could essentially eliminate any useful harvest. Before the delayed effect becomes serious, however, some crop plants with a long latent period might be of value as forage crops.

Adverse effects on progeny from irradiated plants or seed also must be considered. Experiments done with a few species have shown that, depending on the stage of development at which the parent plant was irradiated (see previous discussion), the resultant yield from plants grown from seed of the parent crop may be seriously or moderately affected or not affected at all (see Table 5). Experiments with perennial plants, including various species of trees used as sources of lumber or edible fruits and nuts, have shown that deleterious effects may continue to manifest themselves years after the radiation treatment, particularly in the reproductive system.[64,65]

Influence of Environmental Variables

The main environmental variables known to influence radiation-induced injury in plants are listed in Table 1. Except for dry-seed studies, very few experiments have been done testing the magnitude of effect produced by variation of one or more of these factors in concert with radiation treatment. However, some preliminary results are discussed here.

Lettuce plants given low exposures of radiation show considerably more stimulation of yield early in the growing season under conditions of longer day length than later in the season when the day is shorter.[37] Also, the effects ultimately manifested by perennial plants irradiated during different seasons (while the plants are active or dormant)[12,66,67] or during different photoperiodic stages[68] may differ considerably. The effect of variations in light intensity and temperature on postirradiation survival of *Arabidopsis,* shown in

Fig. 4 Seed yield reduction of five crops after exposure to ^{60}Co gamma radiation at different days after seedling emergence. (a) 'Dayton' barley after exposure to 1 kR at 20 R/min. Maximum reduction was 95% at day 195. Plants irradiated before 130 days after emergence did not survive winter conditions. (b) 'Seneca' wheat after exposure to 1.6 kR at 20 R/min. Maximum reduction was 90% at day 175. Plants irradiated before 85 days after seedling emergence did not survive winter conditions. (c) Maize (WF-9X38-11) after exposure to 2.5 kR at 50 R/min. Maximum reduction was 100% at days 15 to 48. (d) 'Hill' soybeans[99] after exposure to 2.5 kR at 50 R/min. Maximum reduction was 90% at days 6 and 45. (e) Rice (CI 8970-S) after exposure to 25 kR at 50 R/min (redrawn from Siemer et al.[61]). Maximum reduction was 100% at days 37 to 57.

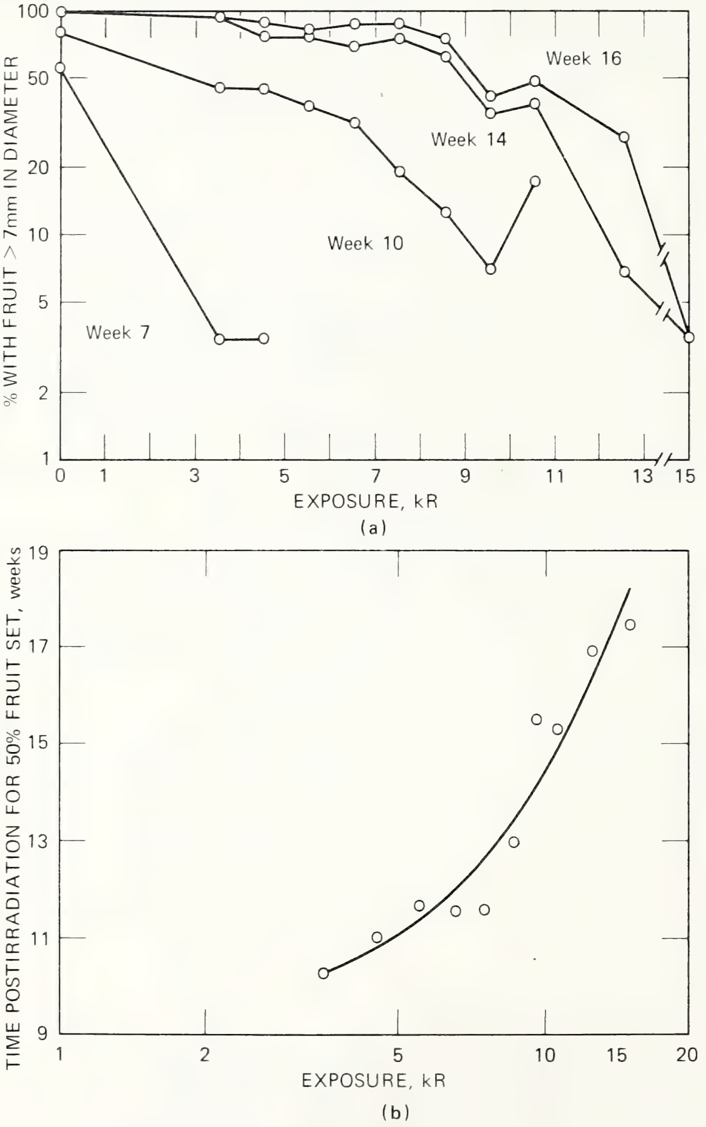

Fig. 5 Data from tomato plants given a 16-hr CR treatment. (a) Percent of plants with fruit vs. exposure at 7, 10, 14 and 16 weeks after irradiation. (b) Postirradiation time in weeks for 50% fruit set vs. exposure.[51]

Table 5

EFFECTS ON YIELD OF THE SUBSEQUENT CROP OF ACUTE GAMMA IRRADIATION DELIVERED AT VARIOUS STAGES OF GROWTH TO SPRING WHEAT, SPRING BARLEY, AND POTATOES[93]

Stage of growth of parent crop when irradiated	Dose, rads	Yield of crop, % of control			
		Grain of spring wheat*		Grain of spring barley*	
		Parent crop	Subsequent crop	Parent crop	Subsequent crop
Two leaf	250	115	93	105	96
	500	97	97	61	103
	1000	68	98	†	
Four leaf	250	98	101	90	101
	500	95	101	50	105
	1000	62	102	†	
Ear emergence	250	86	82	87	96
	500	83	89	59	89
	1000	48	71		
Anthesis	500	91	87	84	86
	1000	73	62	76	47
Postanthesis	500	114	92	85	88
	2000	85	45	89	13
		Potato tubers‡			
		Parent crop	Subsequent crop		
Shoot emergence	2000	51	98		
	4000	15	66		
Stolon formation	2000	74	81		
	4000	33	77		
Tuber initiation	2000	78	96		
	4000	75	54		
	8000	55	†		

*Yield of parent crop figured in grams per plant; yield of subsequent crop in grams per square meter.

†Plants died before maturity.

‡Yield of both crops figured in grams per plant.

Fig. 6, demonstrates that increased temperature is synergistic with radiation treatment in producing deleterious effects.[63] Competition or stress among plants is also known to be a factor in the eventual total effect exhibited by irradiated plants,[69-73] as well as combined effects evident in ecosystem analysis.[18] The maximum difference in effect (a factor of 5) given in Table 2 is considered to be a conservative estimate and may be exceeded in some cases.

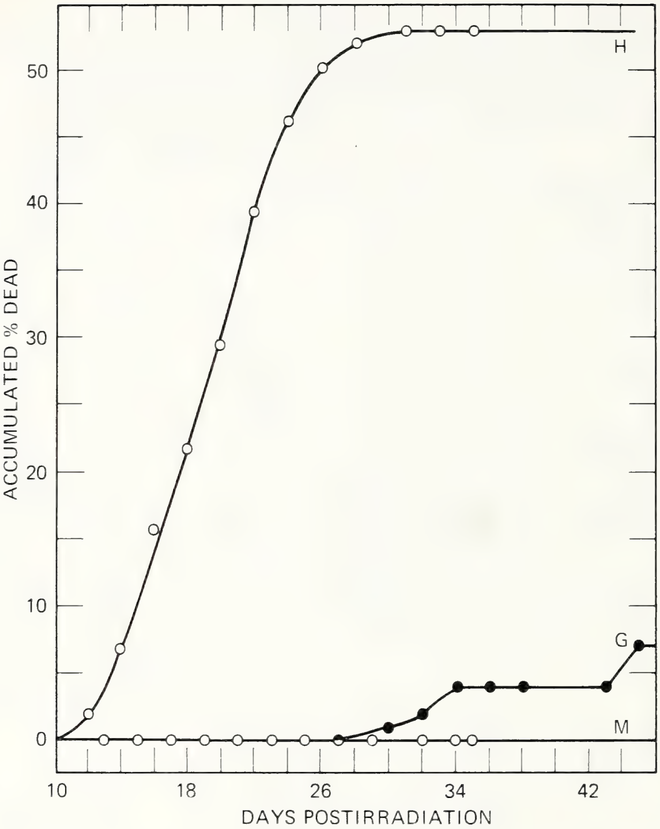

Fig. 6 Relation between accumulated percent dead and number of days postirradiation for plants of *Arabidopsis thaliana* receiving a 16-hr acute gamma exposure to 25 kR and grown under three different sets of conditions of temperature and light: H, 83 to 87°F, full light; G, 68 to 73°F, natural + supplemental light; and M, 68 to 72°F, two-thirds light. Plants were irradiated 13 to 15 days after germination.[63] Maximum percent dead: H, 53%; G, 7%; M, 0%.

DELETERIOUS EFFECTS ON YIELD AND SURVIVAL OF ECONOMIC PLANTS

Although there is a large amount of general information concerning the radiobiological responses of higher plants, there are relatively few published data on deleterious effects on crop yield. The pertinent data available at present are summarized in this section.

Irradiation of Seed Grain, Seed Potato Tubers, Onion Transplants, Bulbs, etc.

The amount of data available for assessing second-generation effects on grain yield from irradiated grain is quite small, though much information exists for other criteria of effect. Radiosensitivities for dry seed of 30 plants of economic value are given in Table 6.[74] However, certain crops not listed, such as peas,

Table 6

LD_{50} (kR) VALUES FOR 30 PLANTS OF ECONOMIC VALUE AFTER ^{60}Co GAMMA IRRADIATION OF DRY SEED*

Common name	Scientific name	Dose rate, R/min	LD_{50}, kR
Alfalfa	*Medicago sativa*	844	38 to 62
Barley	*Hordeum vulgare*	844 to 850	13 to 20
Clover, button	*Medicago orbiculatus*	844	21
Clover, crimson	*Trifolium incarnatum*	844 to 1240	25 to >64
Clover, red	*Trifolium pratense*	795 to 1270	35 to >108
Clover, sweet	*Meliotus* species	844	59
Cowpea	*Vigna sinensis*	1260	11
Dallis grass	*Paspalum dilatatum*	710	>32
Fescue	*Festuca elatior*	844	19
Grape	*Vitis* species	790 to 1240	<4 to <5
Guava	*Psidium guajava*	1240	17
Lespedeza, Korean	*Lespedeza stipulacea*	795	<40
Lupine, blue	*Lupinus angustifolius*	750	>40
Maize	*Zea mays*	840	>15
Millet, German	*Setaria italica*	760	14
Oats	*Avena sativa*	840	17 to 27
Orchard grass	*Dactylis glomerata*	844	11
Papaya	*Carica papaya*	650	12
Peanut	*Arachis hypogea*	1260	10
Pepper	*Capsicum frutescens*	1260	24
Pigeon pea	*Cajanus cajan*	1260	15
Rice	*Oryza sativa*	650 to 1260	<15 to 42
Rye	*Secale cereale*	714 to 840	8 to 16
Sericea	*Lespedeza cuneata*	795 to 840	37 to 46
Sorghum, grain	*Sorghum vulgare*	1260	>40
Soybean	*Glycine max*	1260	11
Tomato	*Lycopersicon esculentum*	609 to 1240	13 to 37
Vetch, hairy	*Vicia villosa*	840	17
Watermelon	*Citrullus vulgaris*	1280	60
Wheat	*Triticum vulgare*	670 to 840	14 to 25

*Modified from Osborne and Lunden.[74]

broad beans, and onions, are much more sensitive than those listed. In a very general way, seed radiosensitivity is related to plant radiosensitivity; i.e., rank order is similar, but actual exposures tolerated are quite different and are highly dependent on moisture content. The least effect on irradiated seeds of cereals is found at moisture contents of about 10 to 13%, and the seeds are more sensitive at moisture contents above or below this level.[75] Depending on the seed moisture content, variations in exposure rate can be as significant for seed irradiation as for irradiation of growing plants but are generally less significant. Seed radiosensitivity is also dependent on the oxygen effect,[76-79] although this is an experimentally induced variable not generally applicable to seed under natural or agricultural conditions.

Exposure of seed potato tubers to 300 R before planting had no effect on yield; 1.2 kR brought about a moderate decrease, and 4.8 kR resulted in a negligible yield.[80] In an experiment using X rays, survival was reduced to 63% by an exposure of 4.0 kR.[81] For small onion transplants an FDS exposure of 2.0 kR resulted in negligible bulb yield; 1.4 kR caused about a 50% reduction; and exposures of 1.1 kR or less produced very little effect on yield (see Table 10). In another study[82] using higher exposure rates, 600 R reduced yield by 28% and 1.0 kR by 78%. Several ornamental bulbs are known to be highly sensitive or are predicted to be from ICV data. Predicted LD_{50} values for FDS exposures are given in Table 7 for a number of species of horticultural interest.

Irradiation of Growing Plants

Because of the significance of stage of development and exposure times or rates on degree of injury produced, it was deemed necessary to specify these details in the summary tables. In many cases only one experiment was performed for a given crop at a specific stage, and in some cases the dosages chosen did not cover the most appropriate range. Also, in most cases the plants were irradiated under laboratory conditions and grown in a greenhouse or growth chamber. Almost no field irradiations have been made. The data on yield and survival have been subjected to computer analysis, which provided estimates (with errors) of the exposures required to reduce yield or survival by about 10, 50, or 90% of unirradiated control. We have used the terms YD_{10}, YD_{50}, or YD_{90} as a shorthand method of specifying the exposure reducing the yield by 10, 50, or 90%, as is usually done for survival, i.e., LD_{50}, etc. It should be emphasized here that it is not only possible but even quite probable that gamma radiation exposures under actual fallout conditions might produce effects greater or less than those indicated in the summary tables (Tables 8 to 12). In other words, these tables can be used only as a general guide to anticipated effects. It is hoped that experiments planned or now under way will greatly improve the accuracy of these tables, at least for some crops. The effects of the beta component of fallout are considered elsewhere.[83] However, if a plant or plant

(Text continues on page 693.)

Table 7

PREDICTED RADIOSENSITIVITIES (36-HR FDS EXPOSURE) OF 25 SPECIES OF ECONOMICALLY IMPORTANT ORNAMENTAL PLANTS GROWN FROM BULBS*

Common name	Scientific name	ICV, μ^3	Predicted LD_{50} ± S.D., kR
Anemone, flame	*Anemone fulgens*	29.2	2.04 ± 0.61
Belladonna lily	*Amaryllis belladonna*	56.5	1.05 ± 0.31
Bluebell, Spanish	*Scilla hispanica*	54.8	1.08 ± 0.32
Crocus	*Crocus* (average of 3 species)	60.8	0.98 ± 0.29
Daffodil	*Narcissus pseudo-narcissus*	63.9	0.93 ± 0.28
Fritillary, checkered	*Fritillaria meleagris*	91.6	0.65 ± 0.19
Gladiolus	*Gladiolus* (average of 4 varieties)	4.7	12.66 ± 3.78
Glory-of-the-snow	*Chiondoxa luciliae*	21.2	2.81 ± 0.84
Grape hyacinth	*Muscari* (average of 2 species)	15.1	3.94 ± 1.18
Hyacinth	*Hyacinthus* (average of 3 varieties)	56.2	1.06 ± 0.32
Lily, Easter	*Lilium longiflorum*	52.2	1.14 ± 0.34
Lily, Formosa	*Lilium formosanum*	66.6	0.89 ± 0.27
Lily, regal	*Lilium regale*	65.4	0.91 ± 0.27
Lily-of-the-valley	*Convallaria majalis*	32.0	1.86 ± 0.56
Mariposa lily	*Calochortus* (average of 2 species)	27.7	2.15 ± 0.64
Narcissus	*Narcissus* (average of 3 species)	39.6	1.50 ± 0.45
Squill, Siberian	*Scilla sibirica*	82.9	0.72 ± 0.21
Star-of-Bethlehem	*Ornithogalum virens*	52.8	1.12 ± 0.34
Tigerflower	*Tigridia pavonia*	17.9	3.32 ± 0.99
Torchlily	*Kniphofia uvaria*	71.2	0.84 ± 0.25
Tritonia (montbretia)	*Tritonia crocata*	8.7	6.84 ± 2.04
Tulip, Darwin	*Tulipa* species	59.8	0.99 ± 0.30
Tulip, Foster (red emperor)	*Tulipa fosteriana*	55.5	1.07 ± 0.32
Tulip, waterlily	*Tulipa kaufmanniana*	32.6	1.83 ± 0.54
Zephyr lily	*Zephyranthes* species	72.9	0.82 ± 0.24

*Used in the general sense, to include bulbs, corms, tubers, and rhizomes.

Table 8 SUMMARY OF RADIOSENSITIVITY DATA FOR CEREALS

Plant	Stage irradiated*	Type of exposure or exposure rate	End point used	Reduction from unirradiated control		
				YD_{10} or LD_{10} ± S.D., R	YD_{50} or LD_{50} ± S.D., R	YD_{90} or LD_{90} ± S.D., R
Spring barley, 'Maris Badger'	Two to four leaf†	30 R/min	Yield (seed weight)	310 ± 120	470 ± 97	890 ± 130
	Ear emergence†	30 R/min	Yield (seed weight)	130 ± 59	620 ± 45	1,950 ± 88
Spring barley, 'Mari'	Seedling‡	FDS	Survival	1580 ± 160	1,990 ± 77	2,400 ± 130
	Seedling‡	FDS	Yield (seed weight)	960 ± 310	1,370 ± 220	2,490 ± 340
	Seedling‡	8-hr CR	Survival	1740 ± 69	1,910 ± 43	2,200 ± 100
Maize, 'Golden Bantam'	Seedling‡	FDS	Survival	2960 ± 140	3,760 ± 85	4,560 ± 140
Maize, WF-9X38-11	Two leaf§	50 R/min	Yield (seed weight)	420 ± 360	800 ± 290	1,840 ± 310
Maize, B14RFx B37RF	Seedling‡	FDS	Survival	4060 ± 100	4,990 ± 80	5,920 ± 140
	Seedling‡	FDS	Yield (seed weight)	4220 ± 250	4,570 ± 190	5,540 ± 210
Spring oat, 'Condor'	Two to four leaf†	30 R/min	Yield (seed weight)	660 ± 220	920 ± 180	1,620 ± 210
	Ear emergence†	30 R/min	Yield (seed weight)	490 ± 110	2,210¶ ± 210	6,920¶ ± 830
	Anthesis†	30 R/min	Yield (seed weight)	420 ± 75	2,210 ± 130	7,090 ± 540
Spring oat, 'Orbit'	Seedling‡	FDS	Survival	2570 ± 160	3,420 ± 170	4,280 ± 290
	Seedling‡	FDS	Yield (seed weight)	1790 ± 30	1,950 ± 30	2,380 ± 20
Rice, CI-8970-S	Panicle emergence§	50 R/min	Yield (seed weight)	2270 ± 1000	14,300 ± 6500	47,200 ± 13,900
Spring wheat, 'Kloka'	Two to four leaf†	30 R/min	Yield (seed weight)	540 ± 46	1,410¶ ± 130	3,800 ± 480
	Ear emergence†	30 R/min	Yield (seed weight)	240 ± 72	900 ± 600	2,730 ± 170
	Anthesis†	30 R/min	Yield (seed weight)	400 ± 100	1,780 ± 140	5,580¶ ± 560
Spring wheat, 'Indus'	Seedling‡	FDS	Survival	2800 ± 110	3,090 ± 72	3,380 ± 110
	Seedling‡	8-hr CR	Survival	2900 ± 170	3,450 ± 110	4,000 ± 210
	Seedling‡	8-hr CR	Yield (seed weight)	1640 ± 330	2,060 ± 270	3,230 ± 410
Winter wheat, 'Capelle'	Ear emergence†	30 R/min	Yield (seed weight)	150 ± 44	860 ± 31	2,800¶ ± 74
	Anthesis†	30 R/min	Yield (seed weight)	320 ± 170	1,560 ± 160	4,980¶ ± 660

*Although meiotic stages are clearly the most sensitive, there is a general lack of data on these stages.

†Communicated by R. S. Russell, Agricultural Research Council, Letcombe Laboratory, England, 1970.

‡A. H. Sparrow and P. J. Bottino, Brookhaven National Laboratory, unpublished data, 1970.

§M. J. Constantin, UT–AEC Agricultural Research Laboratory, unpublished data, 1970.

¶Extrapolated well beyond data points.

Table 9 SUMMARY OF RADIOSENSITIVITY DATA FOR EDIBLE LEGUMES

Plant	Stage irradiated	Type of exposure or exposure rate*	End point	Reduction from unirradiated control		
				YD_{10} or LD_{10} ± S.D., R	YD_{50} or LD_{50} ± S.D., R	YD_{90} or LD_{90} ± S.D., R
Broad bean, 'Sutton'	Vegetative†	30 R/min	Yield (bean weight)	170 ± 84	220 ± 74	350 ± 73
	Flowering†	30 R/min	Yield (bean weight)	51 ± 29	110 ± 25	280 ± 23
Lima bean, 'Fordhook 242'	Seedling‡	FDS	Survival	5450 ± 120	6210 ± 80	6,980 ± 130
	Seedling‡	FDS	Yield (bean weight)	2000 ± 110	2390 ± 90	3,480 ± 50
	Flower bud‡	16-hr CR	Yield (bean weight)		420 ± 430	2,020 ± 460
	Flower and pod‡	16-hr CR	Yield (bean weight)	670 ± 380	1460 ± 470	4,820 ± 560
	Pod‡	16-hr CR	Yield (bean weight)	4350 ± 20	6340 ± 20	11,790 ± 80
	Seedling‡	16-hr CR	Yield (whole plant weight)	3420 ± 450	4190 ± 360	6,280 ± 200
	Seedling‡	16-hr CR	Yield (bean weight)	150 ± 50	920 ± 40	3,020 ± 30
Pea, 'Alaska'	Seedling‡	FDS	Survival	1060 ± 170	2240 ± 71	3,430 ± 120
	Seedling‡	FDS	Yield (whole plant weight)	920 ± 100	1110 ± 88	1,630 ± 65
	Seedling‡	FDS	Yield (pea weight)	800 ± 90	1010 ± 73	1,570 ± 59
	Vegetative†	30 R/min	Yield (pea weight)		380 ± 160	1,060 ± 280
Pea, 'Meteor'	Flowering†	30 R/min	Yield (pea weight)		250 ± 170	600 ± 150
Soybean, 'Hill'	Early blooming§	50 R/min	Yield (bean weight)	550 ± 400	960 ± 350	2,070 ± 280
	Late blooming§	50 R/min	Yield (bean weight)	1160 ± 940	1940 ± 770	4,080 ± 770

*Yield data for other types of exposures are available for lima beans and peas.[39,52]

†Communicated by R. S. Russell, Agricultural Research Council, Letcombe Laboratory, England, 1970.

‡A. H. Sparrow and P. J. Bottino, Brookhaven National Laboratory, unpublished data, 1970.

§M. J. Constantin, UT–AEC Agricultural Research Laboratory, unpublished data, 1970.

Table 10

SUMMARY OF RADIOSENSITIVITY DATA FOR ROOT CROPS

Plant	Stage irradiated	Type of exposure or exposure rate*	End point	Reduction from unirradiated control: YD_{10} or LD_{10} ± S.D., R	YD_{50} or LD_{50} ± S.D., R	YD_{90} or LD_{90} ± S.D., R
Garlic	Bulblets[94]	Acute	Survival	925 ± 265	1,120 ± 195	1,655 ± 300
Onion, 'Yellow Sweet Spanish'	Seedling†	FDS	Survival	1460 ± 57	1,890 ± 45	2,320 ± 86
	Seedling†	FDS	Yield (bulb weight)	1140 ± 74	1,360 ± 50	1,950 ± 76
Potato, 'Majestic'	Shoot emergence‡	80 R/min	Yield (tuber weight)	420 ± 390	1,660 ± 280	5,050 ± 590
	Stolon formation‡	80 R/min	Yield (tuber weight)	1080 ± 710	2,240 ± 590	5,420 ± 650
	Tuber initiation‡	80 R/min	Yield (tuber weight)	970 ± 580	9,330 ± 970	32,200 ± 4290
Radish, 'Cherry Belle'	Seedling†	FDS	Survival	9500 ± 800	12,900 ± 480	16,310 ± 870
	Seedling†	FDS	Yield (root weight)	6840 ± 580	8,870 ± 470	14,420 ± 360
Sugar Beet, 'Sharpes Klein E'	Initiation of swollen hypocotyl‡	80 R/min	Yield (root weight)		1,850 ± 3030	8,400 ± 3710
		80 R/min	Yield (sugar content)	137 ± 1100	1,400 ± 890	4,850 ± 790

*Yield data for other types of exposure are available for onion and radish.[39,50]

†A. H. Sparrow and P. J. Bottino, Brookhaven National Laboratory, 1970.

‡Communicated by R. S. Russell, Agricultural Research Council, Letcombe Laboratory, England, 1970.

Table 11

SUMMARY OF RADIOSENSITIVITY DATA FOR MISCELLANEOUS FRUITS AND VEGETABLES

Plant	Stage irradiated	Type of exposure or exposure rate*	End point	Reduction from unirradiated control YD$_{10}$ or LD$_{10}$ ± S.D., R	YD$_{50}$ or LD$_{50}$ ± S.D., R	YD$_{90}$ or LD$_{90}$ ± S.D., R
Cabbage, 'Ferrys Round Dutch'	Seedling†	FDS	Survival	8,550 ± 530	11,230 ± 300	13,900 ± 490
Lettuce, 'Summer Bibb'	Seedling†	FDS	Survival	4,380 ± 80	4,790 ± 50	5,200 ± 80
	Seedling†	FDS	Yield (whole plant)	4,310 ± 220	4,510 ± 180	5,040 ± 190
	Seedling†	8-hr CR	Survival	4,590 ± 110	5,030 ± 70	5,460 ± 130
	Seedling†	8-hr CR	Yield (whole plant)	3,340 ± 120	4,070 ± 70	6,070 ± 200
Pineapple, 'Smooth Cayenne'	Crown section[95]	Not given	Survival	5,510 ± 990	8,970 ± 850	18,440 ± 780
Spinach, 'Old Dominion'	Seedling†	FDS	Survival	8,410 ± 490	11,800 ± 400	15,100 ± 660
Squash, 'Royal Acorn'	Seedling†	FDS	Survival	4,170 ± 460	6,650 ± 300	9,140 ± 480
	Seedling†	FDS	Yield (whole plant)	3,850 ± 190	6,400 ± 200	
Strawberry, 'Takane'	Stolon[96]	17 R/min	Yield (fruit weight)	1,330 ± 650	6,530 ± 1860	20,800 ± 7300
Tomato, 'Rutgers'	Seedling†	16-hr CR	Survival	11,200 ± 460	13,300 ± 280	15,300 ± 440
	Seedling†	16-hr CR	Yield (fruit weight)	10,100 ± 480	12,100 ± 290	17,600 ± 800

*Yield data for other types of exposures are available for cabbage, lettuce, and squash.[39,50]

†A. H. Sparrow and P. J. Bottino, Brookhaven National Laboratory, 1970.

Table 12

SUMMARY OF RADIOSENSITIVITY DATA FOR PASTURE AND FORAGE CROPS

				Reduction from unirradiated control		
Plant	Stage irradiated	Type of exposure or exposure rate	End point	YD_{10} or LD_{10} ± S.D., R	YD_{50} or LD_{50} ± S.D., R	YD_{90} or LD_{90} ± S.D., R
Meadow fescue	3-week seedling*	30 R/min	Yield (whole plant)	3,030 ± 650	3,710 ± 580	5,570 ± 580
	7-week seedling*	30 R/min	Yield (whole plant)	1,500 ± 1070	2,480 ± 930	5,150 ± 1070
	3- + 7-week seedling*	30 R/min	Yield (whole plant)	2,830 ± 390	3,570 ± 350	5,580 ± 430
Perennial ryegrass	3-week seedling*	30 R/min	Yield (whole plant)		1,590 ± 1200	3,740 ± 1870
	7-week seedling*	30 R/min	Yield (whole plant)		1,930 ± 920	5,080 ± 2200
	3- + 7-week seedling*	30 R/min	Yield (whole plant)	1,090 ± 760	1,920 ± 590	4,170 ± 890
White clover	3-week seedling*	30 R/min	Yield (whole plant)	6,830 ± 1400	11,400 ± 1360	24,000† ± 3980
	7-week seedling*	30 R/min	Yield (whole plant)	6,450 ± 370	23,400 ± 970	69,900† ± 3930
	3- + 7-week seedling*	30 R/min	Yield (whole plant)	6,750 ± 2090	14,000 ± 2580	33,800† ± 9750
White clover, 'White Dutch'	Seedling‡	16-hr CR	Survival	20,300 ± 740	24,200 ± 540	28,100 ± 970
Crested wheatgrass	Seedling[97]	300 R/min	Survival	1,490 ± 850	2000 ± 720	3400 ± 630

*Communicated by R. S. Russell, Agricultural Research Council, Letcombe Laboratory, England, 1970.

†Extrapolated well beyond data points.

‡A. H. Sparrow and P. J. Bottino, Brookhaven National Laboratory, unpublished data, 1970.

gamma radiation required to produce a specified effect will be reduced proportionately.

Herbaceous Species

Cereals (Table 8). Five of the most important cereal crops, which vary appreciably in sensitivity, were studied. For FDS or 8-hr CR exposures to young seedlings, YD_{50} values vary from about 1.4 kR for barley to 4.5 kR for maize, with intermediate values of about 2.0 kR for oats and about 2.1 kR for wheat. No FDS data are available for rice, but for several reasons, it can be expected to be appreciably more resistant than maize. As is generally true, the higher-exposure-rate (30 or 50 R/min) data mostly show greater damage for a given total exposure. For instance, YD_{50} values vary from about 500 to 600 R for barley to 2.2 kR for oats; wheat and maize are intermediate, and rice, by far the most resistant, has a YD_{50} of about 14 kR. At present no yield data exist for three other major cereal crops, namely, rye, sorghum, and pearl millet.

It is known that stage of development at time of exposure influences yield (see previous discussion). Barley and wheat at young-seedling stages are more sensitive than at later stages.[53,54] However, data on lima beans,[52] corn, and rice (Fig. 4) indicate that in these plants meiotic stages are considerably more sensitive than the seedling stage.

We should keep in mind that varietal differences are known to exist for several cereals.[84–86] Differences are generally rather small, but in wheat varietal differences greater than fourfold have been demonstrated.[84]

Legumes (Table 9). So far four different edible legumes (peas, broad beans, lima beans, and soybeans) have been irradiated, and all are highly sensitive or have highly sensitive stages. The YD_{50} for seed yield after FDS seedling irradiation varies from about 1.0 kR for peas to about 3.3 kR for lima beans and, after high-exposure-rate treatments, about 200 R for broad beans. Flower-bud stages are much more sensitive, as shown by both the pea and the lima-bean experiments in which YD_{50} values of approximately 250 and 110 R, respectively, were found following high-dose-rate exposures.

Root Crops (Table 10). The five root crops so far studied vary from an FDS YD_{50} of about 1.4 kR for onions to 8.9 kR for radishes. However, poor texture and bad taste were noted in radishes grown from seedlings irradiated at the higher exposures. Potatoes and sugar beets irradiated at 80 R/min as young plants have YD_{50} values of 1.66 kR and 1.85 kR, respectively. More data are needed for sugar beets since the standard error is large. We should note, however, that reduction in sugar content may be more susceptible to radiation than reduction in root weight, and, as shown in one study, sugar content decreases at a faster rate (R. S. Russell, personal communication), but the decrease does not occur under chronic irradiation.[87] Only survival data are available for garlic; however, the LD_{50} of 1.12 kR indicates that this species is fairly sensitive with regard to yield reduction.

Miscellaneous Fruit and Vegetable Crops (Table 11). Experiments have been conducted with cabbage, lettuce, pineapples, spinach, squash, and tomatoes, but only survival data are available for cabbage, pineapples, and spinach, which have LD_{50} values of approximately 11.2 (FDS), 9.0, and 11.8 (FDS) kR, respectively. The tomato experiment is more difficult to summarize because the YD_{50} was highly dependent on time after irradiation. However, at 10 weeks after exposure, the YD_{50} was approximately 3 kR. Preliminary X-ray experiments with strawberries and raspberries irradiated in the dormant stage indicated only a mild effect on growth at a 16 kR exposure. No yield data were obtained (Sparrow, unpublished). Irradiation of strawberry stolons at 17 R/min produced a YD_{50} of about 6.5 kR. The survival data available for pineapple indicate that crown sections are rather resistant to irradiation, having an LD_{50} of about 9 kR. Limited data for irradiated sugarcane cuttings indicate an LD_{50} of approximately 3 kR.[88]

Pasture and Forage Crops (Table 12). Three grasses and two types of clover have been studied to date. Perennial rye has a YD_{50} of about 1.6 kR and is about one-half as resistant as meadow fescue, which has a YD_{50} of 3.7 kR. White clover, with a YD_{50} of 24 kR, is much more resistant than the grass species at any stage examined. For sweet clover, however, a severe effect (80% reduction) on growth was observed at 4.0 kR after 16-hr acute exposures (Sparrow, unpublished). Only survival data are available for crested wheatgrass and this only at an exposure rate 10 times as high as for white clover and the two grasses.

Woody Species (Fruit, Nut, and Forest Trees, etc.)

Many of the more important forest trees, especially gymnosperms, are extremely sensitive to X or gamma radiation.[9,11,12,26] Recently reported Soviet work has confirmed this high sensitivity by exposing trees to beta irradiation from a number of radionuclides using exposures extending over several years.[89] Brookhaven work showed LD_{50} values for a 16-hr exposure for

Table 13

LD_{50} FOR FIVE SPECIES OF COMMON COMMERCIAL HARDWOODS[11]

Common name	Species	LD_{50} ± S.D., R
Eastern red oak	*Quercus borealis* var. *maxima*	3650 ± 150
Yellow birch	*Betula lutea*	4280 ± 520
Sugar maple	*Acer saccharum*	4720 ± 150
Red maple	*Acer rubrum*	5110 ± 230
White ash	*Fraxinus americana*	7740 ± 260
	Average	5100 ± 700

a number of species to be less than 1.0 kR. Experiments with angiosperms (deciduous trees, including the fruit- and nut-producing trees) have shown them to be more resistant, with LD_{50} values covering a much wider range. LD_{50} data from these experiments are given in Tables 13 and 14 (see also Table 11).

Although all these values are based on actual experimental data, no absolute value of radiosensitivity can be given for any plant species growing under field conditions since a large number of variables influence the amount of injury finally produced by a given exposure. Of particular importance for these woody species is the changing radiosensitivity between the active and dormant stages,[11] the latter being more resistant by a factor of approximately 1.65.

Table 14

AVERAGE LD_{50} FOR EIGHT GENERA (15 SPECIES) OF GYMNOSPERMS[11]

Genera	Number of species	Number of experiments	Range of LD_{50}, R	Average LD_{50} ± S.D., R
Pseudotsuga	1	1		461 ± 71
Pinus	3	3	473 to 818	692 ± 110
Tsuga	1	2	690 to 701	696 ± 6
Picea	4	6	626 to 1186	917 ± 91
Larix	2	2	705 to 834	770 ± 65
Abies	1	1		935 ± 26
Taxus	2	3	475 to 1203	939 ± 233
Thuja	1	1		970 ± 63
All genera	15	19	461 to 1203	826 ± 54

RELATION BETWEEN RADIOSENSITIVITY AND INTERPHASE CHROMOSOME VOLUME

As explained previously, the 36-hr FDS treatment appears to be a reasonable approximation of the exposure regime to which plants would be exposed during postattack fallout. The inverse relation between interphase chromosome volume (ICV) and radioresistance, also referred to previously, is applicable for a 36-hr FDS exposure as well as for shorter exposure times. The regression of ICV vs. LD_{50} for young plants of species of economic value is given in Fig. 1. The regression slope is not significantly different from -1 at the 5% level of significance and is drawn as such. The regression of ICV vs. LD_{10} also has a slope not significantly different from -1 (Fig. 2).

Postirradiation yield and survival data collected for many species of plants indicate a direct relation between LD_{10} and YD_{50} (see Fig. 7 and Table 15). When the data from economic crops only are plotted in this manner (Fig. 3), the

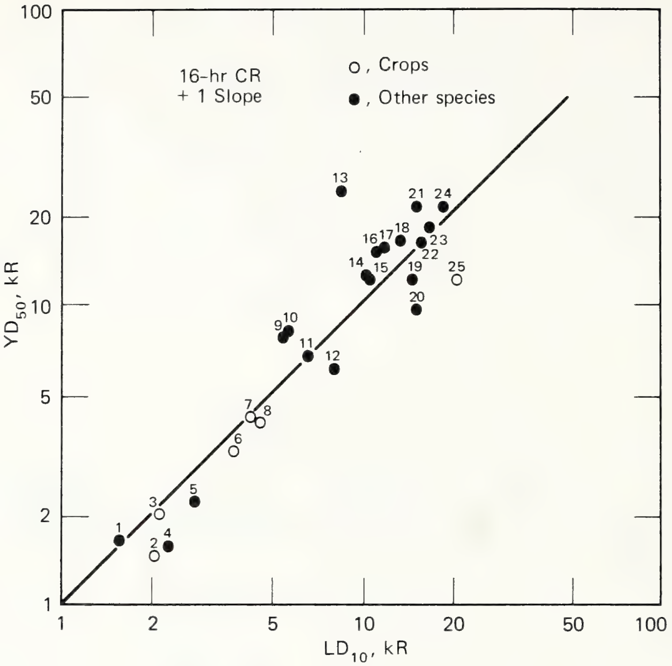

Fig. 7 Log-log regression of YD$_{50}$ against LD$_{10}$ for 25 species of plants given a 16-hr CR exposure as young plants. (See list of species names in Table 15.)

structure is small enough to allow penetration by beta radiation, the amount of regression also fits a +1 slope passing approximately through the origin (0.1, 0.1). Thus the determination of an LD$_{10}$ for any species should provide a fair approximation of the expected YD$_{50}$.

Survival data were collected after 16-hr acute gamma irradiation for 28 species of woody plants, and LD$_{50}$ values were determined. The regression for ICV vs. LD$_{50}$ for these species (both angiosperms and gymnosperms), which has been published,[11] should be used for predictions for woody species since it is appreciably different from the regression for herbaceous plants.

RADIOSENSITIVITY PREDICTIONS (BASED ON ICV DATA)

The regressions described previously were used to predict from ICV measurements the probable sensitivities of many as yet unirradiated plant species.

Predicted YD$_{50}$ values for FDS exposures are given for 89 species of economic crops (Tables 16 and 17). With the exception of rice (Group 7), the cereal crops are concentrated in the four most sensitive groups. Legumes are

Table 15

LIST OF 25 SPECIES OF PLANTS WITH THEIR YD_{50} AND LD_{10} VALUES FOR 16-HR ACUTE GAMMA IRRADIATIONS (AS IN FIG. 7)

No.	Species	YD_{50}, kR	LD_{10}, kR
1	*Haworthia fasciata*	1.65	1.57
2	*Pisum sativum*	1.45	2.03
3	*Hordeum vulgare*	1.80	2.16
4	*Aloe brevifolia*	1.55	2.27
5	*Nigella damascena*	2.20	2.79
6	*Triticum aestivum*	3.11	3.70
7	*Zea mays* (hybrid)	4.20	4.19
8	*Zea mays*	4.00	4.66
9	*Rumex orbiculatus*	7.80	5.38
10	*Cyanotis somaliensis*	8.35	5.64
11	*Chrysanthemum lacustre*	6.75	6.49
12	*Rumex hydrolapathum*	6.00	7.95
13	*Rumex stenophyllus*	24.10	8.32
14	*Rumex aquaticus*	12.80	10.32
15	*Rumex sanguineus*	12.40	10.46
16	*Rumex pulcher*	15.20	11.00
17	*Rumex obtusifolius*	15.30	11.59
18	*Rumex palustris*	16.40	13.04
19	*Rumex maritimus*	12.00	13.17
20	*Rumex confertus*	9.60	14.92
21	*Sedum rupifragum*	21.00	15.00
22	*Rumex conglomeratus*	16.00	16.04
23	*Rumex pseudonatronatus*	17.80	16.54
24	*Rumex crispus*	21.10	18.33
25	*Trifolium repens*	12.25	20.32

distributed over Groups 3 to 6. Root crops are scattered from Groups 2 to 7. Pasture or forage crops are widely distributed from Groups 3 to 8. Numerically, the majority of crop plants have estimated YD_{50} values between 4 and 16 kR. Only seven plants fall above 16 kR, and none of these is a major food crop. Also, the actual sensitivity of one of these (acorn squash) is considerably less than its predicted YD_{50}.[51]

Although few if any data are available on yield reduction for the fruit- and nut-producing trees, it would be expected, as found for herbaceous plants, that the YD_{50} would be appreciably less in each case than the LD_{50}. Predicted LD_{50} values for 16-hr acute exposures for 82 woody plants of economic value (for wood products or for edible fruit and nuts) are given in Table 18. These predictions are based on ICV's from actively growing trees. Trees irradiated while in the dormant stage are somewhat more resistant. However, FDS LD_{50} exposures would be expected to be somewhat less. These predictions are based

(Text continues on page 702.)

Table 16

PREDICTED FALLOUT GAMMA EXPOSURE FOR 50% YIELD REDUCTION (YD_{50}) OF 89 ECONOMIC PLANTS*

Group 1,† <1 kR	Group 2,† 1 to 2 kR	Group 3,† 2 to 4 kR	Group 4,† 4 to 6 kR	Group 5,† 6 to 8 kR	Group 6,† 8 to 12 kR	Group 7, 12 to 16 kR	Group 8, 16 to 20 kR	Group 9, 20 to 24 kR	Group 10, >24 kR
Broad bean	**Barley**	**Bean, lima**	Alfalfa	Asparagus	Alfalfa (vernal)	Brussels sprouts	Bluestem, big	Squash, winter	Okra
Pea	Chives	Cucumber	Bean, kidney	Bluestem, little	Artichoke, globe	Dallis grass	Peppermint	Squash, zucchini	Rape, winter
	Garlic	Fescue, reed	Brome, smooth	Buckwheat	Bean, mung	Flax	Squash, butternut		
	Leek	**Maize** (hybrid)	Fescue, sheep	Cauliflower	Beet	Grama, blue			
	Lentil	**Oats**	Hops	Celery	Broccoli	Mustard, India			
	Onion	Pea, field	**Lettuce**	Cotton	Cabbage, Chinese	Potato, sweet			
	Rye	Ryegrass, perennial	Millet, pearl	Cowpea	Cantaloupe	Rice			
		Spinach, round seed	Orchard grass	Dill	Carrot	Spearmint			
		Wheat	Peanut	Kale	Castor bean	Strawberry			
		Wheatgrass, bearded	Rhubarb	Muskmelon	Clover, red	Turnip			
		Wheatgrass, crested	Safflower	Parsnip	Eggplant				
			Sorghum	Pepper, bush red (Bell)	Mustard, black				
			Sunflower	**Squash, acorn**	Mustard, white				
			Wheatgrass	Sugarcane	Parsley				
				Swiss chard	**Radish**				
				Timothy	Rape, bird				
				Tobacco, common	Rutabaga				
				Watermelon	Sesame, oriental				
					Sisal hemp				
					Soybean				

*Predictions are for young plants and are based on a regression of ICV vs. YD_{50} for seven species of economic plants given a 36-hr FDS exposure series.

†Boldface type indicates these species for which actual FDS data are available. These data have been used to place the species in their appropriate groups.

Table 17 SCIENTIFIC AND COMMON NAMES FOR 89 ECONOMIC PLANTS FOR WHICH YD_{50} PREDICTIONS HAVE BEEN MADE*

Scientific name	Common name†
Group 1 (<1 kR)	
Pisum sativum	**Pea**
Vicia faba	Broad bean
Group 2 (1 to 2 kR)	
Allium cepa	**Onion**
Allium porrum	Leek
Allium sativum	Garlic
Allium schoenoprasum	Chives
Hordeum vulgare	**Barley**
Lens culinaris	Lentil
Secale cereale	Rye
Group 3 (2 to 4 kR)	
Agropyron cristatum	Crested wheatgrass
Agropyron trachycaulum	Bearded wheatgrass
Avena sativa	**Oats**
Cucumis sativus	Cucumber
Festuca elatior	Reed fescue
Lolium perenne	Perennial ryegrass
Phaseolus limensis	**Lima bean**
Pisum sativum arvense	Field pea
Spinacia oleracea	Round seed spinach
Triticum aestivum	**Wheat**
Zea mays	**Maize**
Group 4 (4 to 6 kR)	
Agropyron intermedium	Wheatgrass
Arachis hypogaea	Peanut
Bromus inermis	Smooth brome
Carthamus tinctorius	Safflower
Dactylis glomerata	Orchard grass
Festuca ovina	Sheep fescue
Helianthus annuus	Sunflower
Humulus lupulus	Hops
Lactuca sativa	**Lettuce**
Medicago sativa	Alfalfa
Pennisetum glaucum	Pearl millet
Phaseolus vulgaris	Kidney bean
Rheum rhaponticum	Rhubarb
Sorghum vulgare	Sorghum
Group 5 (6 to 8 kR)	
Andropogon scoparius	Little bluestem
Anethum graveolens	Dill
Apium graveolens	Celery
Asparagus officinalis	Asparagus
Beta cicla	Swiss chard
Brassica oleracea var. *acephala*	Kale
Brassica oleracea var. *botrytis*	Cauliflower
Capsicum frutescens	Bush red pepper (Bell)
Citrullus vulgaris	Watermelon
Cucumis melo	Muskmelon
Cucurbita pepo	**Acorn squash**
Fagopyrum sagittatum	Buckwheat
Gossypium hirsutum	Cotton
Group 5 (6 to 8 kR) (continued)	
Nicotiana tabacum	Common tobacco
Pastinaca sativa	Parsnip
Phleum pratense	Timothy
Saccharum officinarum	Sugarcane
Vigna sinensis	Cowpea
Group 6 (8 to 12 kR)	
Agave rigida	Sisal hemp
Beta vulgaris	Beet
Brassica campestris	Bird rape
Brassica hirta	White mustard
Brassica napobrassica	Rutabaga
Brassica nigra	Black mustard
Brassica oleracea var. *italica*	Broccoli
Brassica pekinensis	Chinese cabbage
Cucumis melo var. *cantalupensis*	Cantaloupe
Cynara scolymus	Globe artichoke
Daucus carota var. *sativa*	Carrot
Glycine max	Soybean
Medicago sativa	Vernal alfalfa
Petroselinum crispum	Parsley
Phaseolus aureus	Mung bean
Raphanus sativus	**Radish**
Ricinus communis	Castor bean
Sesamum indicum	Oriental sesame
Solanum melongena	Eggplant
Trifolium pratense	Red clover
Group 7 (12 to 16 kR)	
Bouteloua gracilis	Blue grama
Brassica juncea	Indian mustard
Brassica oleracea var. *gemmifera*	Brussels sprouts
Brassica rapa	Turnip
Fragaria species	Strawberry
Ipomoea batatas	Sweet potato
Linum usitatissimum	Flax
Mentha spicata	Spearmint
Oryza sativa	Rice
Paspalum dilatatum	Dallis grass
Group 8 (16 to 20 kR)	
Andropogon gerardi	Big bluestem
Cucurbita moschata 'Butternut'	Butternut squash
Mentha piperita	Peppermint
Group 9 (20 to 24 kR)	
Cucurbita maxima	Winter squash
Cucurbita pepo var. *medullosa*	Zucchini squash
Group 10 (>24 kR)	
Brassica napus	Winter rape
Hibiscus esculentus	Okra

*See Table 16.

†Boldface type indicates those species for which actual FDS data are available. These data have been used to place the species in their appropriate groups.

Table 18

PREDICTED 16-HR (ACUTE) GAMMA LD$_{50}$ EXPOSURES
FOR 82 WOODY PLANTS OF ECONOMIC VALUE[11]

Common name	Scientific name	LD$_{50}$ ± S.D., kR
Almond	*Prunus amygdalus* Batsch 'Nonpareil'	3.11 ± 1.16
Apple, common	*Pyrus malus* L. 'Northern Spy'	4.60 ± 1.75
Apricot	*Prunus armeniaca* L. 'Blenheim'	3.00 ± 1.12
Arborvitae, eastern	*Thuja occidentalis* L.	1.50 ± 0.55
Arborvitae, giant	*Thuja plicata* Donn	1.70 ± 0.63
Ash, white	*Fraxinus americana* L.	7.11 ± 2.77
Aspen, quaking	*Populus tremuloides* Michx.	4.80 ± 1.83
Avocado, American	*Persea americana* Mill.	2.81 ± 1.05
Beech, American	*Fagus grandifolia* Ehrh.	6.41 ± 2.48
Birch, yellow	*Betula lutea* Michx. f.	6.63 ± 2.57
Blueberry, highbush	*Vaccinium corymbosum* L.	5.54 ± 2.13
Blueberry, lowbush	*Vaccinium angustifolium* Ait.	5.85 ± 2.25
Buckeye, yellow	*Aesculus octandra* Marsh.	7.11 ± 2.77
Cassava	*Manihot dulcis* Pax 'Valenca'	3.50 ± 1.32
Cedar, eastern red	*Juniperus virginiana* L.	1.35 ± 0.50
Cedar-of-Lebanon	*Cedrus libani* Loud.	0.84 ± 0.31
Cherry, mazzard	*Prunus avium* L. 'Windsor'	3.60 ± 1.36
Cherry, sour	*Prunus* X *cerasus* L.	5.85 ± 2.25
Chestnut, American	*Castanea dentata* (Marsh.) Borkh.	3.77 ± 1.42
Cranberry	*Vaccinium macrocarpon* Ait.	6.41 ± 2.48
Cryptomeria	*Cryptomeria japonica* D. Don 'Araucarioides'	1.22 ± 0.45
Cypress, bhutan	*Cupressus duclouxiana* Hickel	1.58 ± 0.58
Cypress, common bald	*Taxodium distichum* (L.) Rich.	1.71 ± 0.63
Eucalyptus, messmate stringy bark	*Eucalyptus obliqua* L' Her.	3.00 ± 1.12
Fig, common	*Ficus carica* L. 'Celeste'	6.21 ± 2.40
Fir, alpine	*Abies lasiocarpa* (Hook.) Nutt.	0.62 ± 0.23
Fir, balsam	*Abies balsamea* (L.) Mill.	0.75 ± 0.28
Fir, Douglas	*Pseudotsuga douglasii* Carr.	0.99 ± 0.37
Fir, grand	*Abies grandis* Lindl.	0.62 ± 0.23
Fir, white	*Abies concolor* Hoopes	0.81 ± 0.30
Grape	*Vitis* species 'Concord'	5.85 ± 2.25
Grape	*Vitis* species 'Delaware'	5.69 ± 2.19
Grapefruit	*Citrus paradisi* Macf.	3.27 ± 1.23
Hemlock, Canada	*Tsuga canadensis* (L.) Carr.	0.72 ± 0.27
Hemlock, Pacific	*Tsuga heterophylla* Sarg.	0.80 ± 0.30
Hickory, bitternut	*Carya cordiformis* (Wang.) K. Koch	7.69 ± 3.01
Hickory, mockernut	*Carya tomentosa* (Poir.) Nutt.	7.69 ± 3.01
Hickory, shagbark	*Carya ovata* (Mill.) K. Koch	6.03 ± 2.32
Hickory, shellbark	*Carya laciniosa* (Michx. f.) Loud.	4.10 ± 1.55
Juniper, common	*Juniperus communis* L.	1.49 ± 0.55
Larch, eastern	*Larix laricina* (Duroi) K. Koch	0.69 ± 0.26
Larch, European	*Larix decidua* Mill.	0.77 ± 0.29
Larch, Japanese	*Larix leptolepis* Gord.	0.85 ± 0.31
Larch, western	*Larix occidentalis* Nutt.	0.85 ± 0.31

Table 18 (Continued)

Common name	Scientific name	LD_{50} ± S.D., kR
Lemon	*Citrus limonia* Burm. f. 'Villa Franca'	4.18 ± 1.58
Linden, American	*Tilia americana* L.	6.03 ± 2.32
Locust, black	*Robinia pseudoacacia* L.	3.15 ± 1.18
Maple, sugar	*Acer saccharum* Marsh.	4.80 ± 1.79
Oak, blackjack	*Quercus marilandica* Muenchh.	3.45 ± 1.30
Oak, eastern red	*Quercus borealis* Michx. f. var. *maxima* (Marsh.) Ashe	3.36 ± 1.26
Oak, post	*Quercus stellata* Wang.	3.96 ± 1.50
Oak, swamp chestnut	*Quercus prinus* L.	3.11 ± 1.16
Oak, white	*Quercus alba* L.	2.93 ± 1.10
Orange, mandarin	*Citrus reticulata* Blanco 'Cleopatra'	4.91 ± 1.87
Orange, sweet	*Citrus sinensis* Osbeck 'Parson Brown'	4.18 ± 1.58
Peach	*Prunus persica* (L.) Patsh.	4.60 ± 1.75
Pecan	*Carya illinoensis* (Wang.) K. Koch 'Sioux'	2.90 ± 1.08
Pine, Austrian	*Pinus nigra* Arnold	0.61 ± 0.23
Pine, eastern white	*Pinus strobus* L. 'Pendula'	0.52 ± 0.20
Pine, Himalayan	*Pinus griffithii* McClel.	0.50 ± 0.19
Pine, Japanese red	*Pinus densiflora* Sieg. et Zucc. 'Umbraculifera'	0.60 ± 0.22
Pine, loblolly	*Pinus taeda* L.	0.63 ± 0.23
Pine, ponderosa	*Pinus ponderosa* Dougl.	0.58 ± 0.22
Pine, pitch	*Pinus rigida* Mill.	0.67 ± 0.25
Pine, red	*Pinus resinosa* Ait.	0.70 ± 0.26
Pine, Scotch	*Pinus sylvestris* L.	0.62 ± 0.23
Pine, shore	*Pinus contorta* Loud.	0.70 ± 0.26
Pine, slash	*Pinus caribaea* Morelet	0.77 ± 0.29
Pine, sugar	*Pinus lambertiana* Dougl.	0.41 ± 0.16
Pine, Virginia	*Pinus virginiana* Mill.	0.69 ± 0.26
Plum, garden	*Prunus domestica* L.	4.60 ± 1.75
Redwood	*Sequoia sempervirens* Endl.	1.46 ± 0.54
Sequoia, giant	*Sequoiadendron giganteum* Buchholz	1.72 ± 0.64
Spruce, black	*Picea mariana* (Mill.) BSP.	0.84 ± 0.31
Spruce, Colorado	*Picea pungens* Engelm.	0.76 ± 0.28
Spruce, engelmann	*Picea engelmanni* Parry	0.73 ± 0.27
Spruce, Norway	*Picea abies* (L.) Karst.	0.73 ± 0.27
Spruce, red	*Picea rubens* Sarg.	0.57 ± 0.21
Spruce, white	*Picea glauca* (Moench) Voss	0.77 ± 0.29
Walnut, eastern black	*Juglans nigra* L.	3.83 ± 1.45
Walnut, Persian	*Juglans regia* L.	4.80 ± 1.83
Yew, Canada	*Taxus canadensis* Marsh.	0.99 ± 0.36

on the effective exposure dose actually received by the plants. Partial shielding by soil, water, or other plants, which has not been considered, could be important in certain instances.

Predicted values of LD_{50} (in kiloroentgens) for the dominant woody species in the major types of ecosystems are given in Table 19. A fallout gamma exposure of 1 to 2 kR would be expected to virtually eliminate the productive capacity of coniferous forests during a season of active growth and might seriously affect the natural balance of species in other forests and thus increase the possibility of secondary damage from fire, flood, and loss of nutrients.

It must be remembered that these predictions are made from regressions based on experiments handled under specific conditions. Any of a number of variables (see previous discussion of variables) can alter the predicted values in either direction and thus must be taken into account in planning experiments, evaluating radiobiological data, or assessing damage.

SUMMARY AND CONCLUSIONS

1. Problems are encountered in trying to anticipate the degree of damage to native or cultivated plants which would be produced by the radiation released by high-level fallout from nuclear detonations. Since almost no pertinent data resulting from such a disaster exist, it is necessary to extrapolate from other radiobotanical data. For short-term consideration of postattack damage, the gross radiation effects of greatest economic importance are reduced vegetative growth, reduced yield, and plant deaths. Alterations in normal plant tolerance to environmental stress also may occur, as may secondary effects resulting from the death of plants, such as loss of nutrients and increased probability of flood or fire damage. Although plants receiving postattack fallout would be exposed to both beta and gamma radiation, this report discusses mainly the results expected from the latter. Present data indicate an RBE of about 1 for these two radiations, but distribution and depth dose is a problem with beta radiation. The possibility of an interaction between the two types of radiation injury is very real and must be considered.

2. There is a wide range in radiosensitivities of plants determined or influenced by many biological, radiological, and environmental factors. Variation in exposure rate is an important factor, high rates being generally more effective (by a factor as great as 4) than low rates in reducing survival and yield. Variation in radiosensitivity among species exceeds a factor of 100. Within a species the stage of development of the plant may also affect its sensitivity by as much as a factor of 50; seeds are most resistant, and certain stages of meiosis are most sensitive (Table 4). Hence the seasonal timing of the exposure is an important variable.

3. Among the most important environmental conditions influencing the radiation response are temperature, light, and competition (Table 1). Experi-

Table 19

PREDICTED SENSITIVITY TO GAMMA RADIATION OF MAJOR WOODY ECOSYSTEMS AND THEIR DOMINANT PLANT SPECIES

Major ecosystem and vegetation type	**Dominant species**	**Common name***	**Predicted† 16-hr acute gamma LD_{50} ± S.D., kR**
	Coniferous Forests		
Boreal	*Abies balsamea*	Balsam fir	0.89 ± 0.03‡
	Picea glauca	White spruce	0.85 ± 0.05‡
Subalpine (Rocky Mts.)	*Abies lasiocarpa*	Alpine fir	0.62 ± 0.23
	Picea engelmanni	Engelmann spruce	0.73 ± 0.27
Montane (Rocky Mts.)	*Pinus ponderosa*	Ponderosa pine	0.58 ± 0.22
	Pseudotsuga douglasii	Douglas fir	0.99 ± 0.37
Sierra Cascades	*Abies concolor*	White fir	0.81 ± 0.30
	Pinus jeffreyi	Jeffrey pine	0.67 ± 0.25
	Pinus lambertiana	Sugar pine	0.41 ± 0.16
	Pinus ponderosa	Ponderosa pine	0.58 ± 0.22
	Pseudotsuga douglasii	Douglas fir	0.46 ± 0.07‡
Pacific conifer	*Abies grandis*	Grand fir	0.62 ± 0.23
	Thuja plicata	Giant arborvitae	1.70 ± 0.63
	Tsuga heterophylla	Western hemlock	0.80 ± 0.30
	Deciduous Forests		
Mixed mesophytic	*Acer saccharum*	Sugar maple	4.80 ± 1.79
	Fagus grandifolia	American beech	6.41 ± 2.48
	Liriodendron tulipifera	Yellow poplar	3.00 ± 1.12
	Magnolia acuminata	Cucumbertree magnolia	3.71 ± 1.40
	Quercus alba	White oak	2.93 ± 1.10
	Tilia americana	American linden	6.03 ± 2.32
Beech–maple	*Acer saccharum*	Sugar maple	4.80 ± 1.79
Maple–basswood	*Fagus grandifolia*	American beech	6.41 ± 2.48
	Tilia americana	American linden	6.03 ± 2.32
	Tsuga canadensis	Canada hemlock	0.72 ± 0.27
Hemlock–hardwood	*Acer saccharum*	Sugar maple	4.72 ± 0.15‡
	Betula lutea	Yellow birch	4.28 ± 0.52‡
	Pinus resinosa	Red pine	0.78 ± 0.03‡
	Pinus strobus	Eastern white pine	0.47 ± 0.01‡
	Tsuga canadensis	Canada hemlock	0.70 ± 0.05‡
Oak–chestnut	*Castanea dentata*	American chestnut	3.77 ± 1.42
	Pinus rigida	Pitch pine	0.67 ± 0.25
	Quercus coccinea	Scarlet oak	4.60 ± 1.75
	Quercus prinus	Swamp oak	3.11 ± 1.16
Oak–hickory	*Carya cordiformis*	Bitternut hickory	7.69 ± 3.01
	Carya laciniosa	Shellbark hickory	4.10 ± 1.55
	Carya ovata	Shagbark hickory	6.03 ± 2.32
	Carya tomentosa	Mockernut hickory	7.69 ± 3.01
	Pinus taeda	Loblolly pine	0.63 ± 0.23
	Quercus alba	White oak	2.93 ± 1.10
	Quercus borealis var. *maxima*	Eastern red oak	3.36 ± 1.26
	Quercus marilandica	Blackjack oak	3.45 ± 1.30
	Quercus stellata	Post oak	3.96 ± 1.50
	Quercus velutina	Black oak	5.02 ± 1.92

*From *Standardized Plant Names.*[98]

†Based on calculations of ICV from active meristems.

‡Observed mortality in actual experiments.[11]

mental data suggest that these factors could cause the response to vary by as much as a factor of 10, excluding the effects of drought. Since significant exposures to radiation can be expected to delay flower initiation and ripening, plants with a growing season clearly limited by conditions of climate (e.g., tomato) might survive through the growing season but would produce essentially no useful yield. Also, since different species die at different rates after lethal irradiation, plants that die very quickly would be virtually worthless under postattack conditions, but those dying more slowly might be of some limited value.

4. The viability and vigor of seed from irradiated plants must be considered. The seed is used to produce the next crop, and adverse effects are sometimes present even in seed that superficially looks perfectly normal. Also, deleterious effects on growth or yield may be manifested in the subsequent crop (for annuals), and in some cases these effects appear several years after exposure (for perennials), especially in the reproductive processes.

5. It is useful to have a yield end point that can be compared for all crops, e.g., the exposure required to reduce yield by 50% (YD_{50}). Because seeds are generally relatively resistant compared with growing plants of the same species, they probably would not present a problem while in storage. The seedling LD_{50} for irradiated dry seeds is above 10 kR for most but not all crop plants (Table 6). Other propagules, such as seed potato tubers and small onion transplants, are more sensitive, having a YD_{50} value of about 1.5 kR.

6. A brief summary of existing sensitivity data on crop and other cultivated plants follows. (Keep in mind that modifying factors can have a large effect on response and there is no such thing as an absolute value under field conditions.) Most of the small grain cereals are relatively sensitive, having YD_{50} exposures that range from 0.5 to 5.0 kR (Table 8). Rice is an exception, having a YD_{50} of about 14.0 kR for young seedlings. Edible legumes vary more in sensitivity; exposures of 220 R to about 6.0 kR to vegetative stages produce a YD_{50} (Table 9). For flowering stages YD_{50} values range between about 100 and 400 R. The YD_{50} values for root crops vary from 1.4 kR for onions to about 9.0 kR for radishes; potatoes and sugar beets are intermediate in sensitivity (Table 10). The miscellaneous crops (in order of increasing resistance: lettuce, pineapple, strawberry, squash, spinach, cabbage, and tomato) have YD_{50} or LD_{10} values ranging from 4.5 to 12 kR (Table 11). The pasture and forage crop plants have a very wide range in sensitivity; YD_{50} or LD_{10} values range from about 1.5 to 23 kR (Table 12). Finally, some woody plants of economic importance are highly sensitive. The gymnosperms studied have an average LD_{50} value of 826 R (Table 14). The deciduous trees are somewhat more resistant, generally having LD_{50} values ranging from 3.6 to 7.7 kR (Table 13). The exposures seriously affecting their economic usefulness would be much lower.

7. The inverse relation between ICV and radioresistance holds for simulated-fallout-decay gamma exposures as reported previously for acute and chronic

gamma exposures. A regression of YD_{50} vs. LD_{10} has a slope not significantly different from +1 (Figs. 3 and 7). Thus an LD_{10} for any species can be used as a fair approximation of the YD_{50}. A table of YD_{50} predictions based on ICV is given for 89 species of economic plants showing the distribution of various crop plants over the entire range of sensitivity from less than 1 to more than 24 kR (Tables 16 and 17). Predictions of LD_{50} are also given for 25 species of ornamental plants (Table 7) and 82 species of woody plants (Table 18).

8. These predictions of survival and yield, although based on a large amount of experimental data, are for stated experimental conditions and average environmental conditions. They can be expected to vary considerably under actual fallout conditions and should not be considered absolute for any species. However, they should be useful in damage-assessment work because they give some advance indication of which crops might survive at various radiation levels and be available for human and/or animal consumption. For example, most small grain cereals (not including maize and rice) would be virtually useless where fallout gamma exposures exceed about 2.0 kR. Therefore only fields in fringe areas or away from the main fallout patterns would produce normal yields.

9. Finally, we should point out that the amount of radiobiological data is still highly inadequate to permit confident predictions of expected responses of many important species from high-level fallout-gamma exposure. This is especially true for yield and is even more critical if exposure occurs during meiosis or development of reproductive structures. Inadequate information about beta-radiation injury and possible interaction or synergism between beta and gamma radiation further complicates the problem. Clearly a much greater research effort is needed to fill the gaps in our radiobiological knowledge of economically important plant species. (See also the recommendations of the various working groups of this symposium, especially those concerned with the vulnerability of crops to beta and gamma radiation.[100,101])

ACKNOWLEDGMENTS

This research was carried out at Brookhaven National Laboratory under the auspices of the U. S. Atomic Energy Commission.

We wish to thank J. Bryant, Brenda Floyd, E. E. Klug, Anne F. Nauman, Virginia Pond, Leanne Puglielli, L. A. Schairer, and Pamela Silimperi for technical assistance and K. H. Thompson for statistical analyses.

We gratefully acknowledge the use of unpublished data from several sources. These data are acknowledged in the appropriate tables. Recognition of the need for this review resulted from discussions at previous conferences sponsored by the Office of Civil Defense.

REFERENCES

1. J. P. Witherspoon and A. K. Corney, Differential and Combined Effects of Beta, Gamma and Fast Neutron Irradiation of Soybean Seedlings, *Radiat. Bot.,* **10**: 429–435 (1970).
2. R. S. Bruce and R. S. Russell, Agricultural Aspects of Acute and Chronic Contamination Situations, in *Environmental Contamination by Radioactive Materials,* Seminar Proceedings, Vienna, 1969, pp. 79–89, International Atomic Energy Agency, Vienna, 1969 (STI/PUB/226).
3. S. A. Griffin and G. R. Eisele, Problems in Postattack Livestock Salvage, this volume.
4. Y. C. Ng and H. A. Tewes, Radionuclide Body Burdens and Hazards from Ingestion of Foodstuffs Contaminated by Fallout, this volume.
5. R. S. Russell (Ed.), *Radioactivity and Human Diet,* Pergamon Press, Inc., New York, 1966.
6. R. S. Russell, B. O. Bartlett, and R. S. Bruce, The Significance of Long-Lived Nuclides After a Nuclear War, this volume.
7. J. E. Gunckel, Modifications of Plant Growth and Development Induced by Ionizing Radiations, in *Encyclopedia of Plant Physiology,* Vol. 15, Part 2, pp. 365–387, Springer-Verlag, Berlin, 1961.
8. J. E. Gunckel and A. H. Sparrow, Ionizing Radiations: Biochemical, Physiological and Morphological Aspects of Their Effects on Plants, in *Encyclopedia of Plant Physiology,* Vol. 16, pp. 555–611, Springer-Verlag, Berlin, 1961.
9. A. H. Sparrow, Research Uses of the Gamma Field and Related Radiation Facilities at Brookhaven National Laboratory, *Radiat. Bot.,* **6**: 377–405 (1966).
10. A. H. Sparrow, J. P. Binnington, and V. Pond, Bibliography on the Effects of Ionizing Radiations on Plants 1896–1955, USAEC Report BNL-504, Brookhaven National Laboratory, July 1958.
11. A. H. Sparrow, A. F. Rogers, and S. S. Schwemmer, Radiosensitivity Studies with Woody Plants. I. Acute Gamma Irradiation Survival Data for 28 Species and Predictions for 190 Species, *Radiat. Bot.,* **8**: 149–186 (1968).
12. A. H. Sparrow, S. S. Schwemmer, E. E. Klug, and L. Puglielli, Radiosensitivity Studies with Woody Plants. II. Survival Data for 13 Species Chronically Irradiated (up to 8 Years), *Radiat. Res.,* **44**: 154–177 (1970).
13. I. M. Vasil'ev, *Effect of Ionizing Radiations on Plants,* USAEC Report AEC-tr-5836, translated by Lydia Venters from Deistvie Ioniziruyushchikh Izlucheniina Rasteniya, Izdatel'stvo Akademii Nauk SSSR, Moscow, 1962.
14. G. M. Woodwell and A. H. Sparrow, Effects of Ionizing Radiation on Ecological Systems, in *Ecological Effects of Nuclear War,* G. M. Woodwell (Ed.), USAEC Report BNL-917, pp. 20–38, Brookhaven National Laboratory, August 1965.
15. J. L. Brewbaker and G. C. Emery, Pollen Radiobotany, *Radiat. Bot.,* **1**: 101–154 (1962).
16. A. H. Sparrow, L. A. Schairer, and R. C. Sparrow, Relationship Between Nuclear Volumes, Chromosome Numbers and Relative Radiosensitivities, *Science,* **141**: 163–166 (1963).
17. G. M. Woodwell and J. K. Oosting, Effects of Chronic Gamma Irradiation on the Development of Old Field Plant Communities, *Radiat. Bot.,* **5**: 205–222 (1965).
18. G. M. Woodwell and A. H. Sparrow, Predicted and Observed Effects of Chronic Gamma Radiation on a Near-Climax Forest Ecosystem, *Radiat. Bot.,* **3**: 231–237 (1963).
19. K. Yamakawa and A. H. Sparrow, Correlation of Interphase Chromosome Volume and Reduction of Viable Seed Set by Chronic Irradiation of 21 Cultivated Plants During Reproductive Stages, *Radiat. Bot.,* **5**: 557–566 (1965).

20. K. Yamakawa and A. H. Sparrow, The Correlation of Interphase Chromosome Volume with Pollen Abortion Induced by Chronic Gamma Irradiation, *Radiat. Bot.*, **6**: 21–38 (1966).
21. A. H. Sparrow, Radiation Sensitivity of Cells During Mitotic and Meiotic Cycles with Emphasis on Possible Cytochemical Changes, *Ann. N. Y. Acad. Sci.*, **51**: 1508–1540 (1951).
22. A. H. Sparrow and V. Pond, Some Cytogenetic and Morphogenetic Effects of Ionizing Radiation on Plants, in *Proceedings of a Conference on Radioactive Isotopes in Agriculture*, Jan. 12–14, 1956, East Lansing, Mich., USAEC Report TID-7512, pp. 125–139, Argonne National Laboratory, January 1956.
23. N. Nybom, Some Further Experiments on Chronic Gamma Irradiation of Plants, *Botan. Notiser*, **109**: 1–11 (1956).
24. A. K. Sharma and A. K. Chatterji, Chromosome Size as a Factor in Radiosensitivity, *Nucleus (Calcutta)*, **5**: 67–74 (1962).
25. A. H. Sparrow, The Role of the Cell Nucleus in Determining Radiosensitivity, Brookhaven Lecture Series No. 17, USAEC Report BNL-766, Brookhaven National Laboratory, May 16, 1962.
26. A. H. Sparrow, Comparisons of the Tolerances of Higher Plant Species to Acute and Chronic Exposures of Ionizing Radiation, *Jap. J. Genet., Suppl.*, **40**: 12–37 (1965).
27. A. H. Sparrow, R. L. Cuany, J. P. Miksche, and L. A. Schairer, Some Factors Affecting the Responses of Plants to Acute and Chronic Radiation Exposures, *Radiat. Bot.*, **1**: 10–34 (1961).
28. A. H. Sparrow, R. C. Sparrow, K. H. Thompson, and L. A. Schairer, The Use of Nuclear and Chromosomal Variables in Determining and Predicting Radiosensitivities, *Radiat. Bot., Suppl.*, **5**: 101–132 (1965).
29. S. C. Sur, Interphase Chromosome Volume-Sensitivity Relationship in Some Diploid Annuals, *Technology (Sindri)*, **5**: 208–212 (1968).
30. S. A. Gordon, The Effects of Ionizing Radiation on Plants: Biochemical and Physiological Aspects, *Quart. Rev. Biol.*, **32**: 3–14 (1957).
31. International Atomic Energy Agency, Effects of Low Doses of Radiation on Crop Plants, Technical Report Series, No. 64, Vienna, 1966 (STI/DOC/10/64).
32. K. Sax, The Stimulation of Plant Growth by Ionizing Radiation, *Radiat. Bot.*, **3**: 179–186 (1963).
33. J. Skok, W. Chorney, and E. J. Rakosnik, Jr., An Examination of Stimulatory Effects of Ionizing Radiation in Plants, *Radiat. Bot.*, **5**: 281–292 (1965).
34. A. I. Grechushnikov, V. S. Serebrenikov and V. P. Kiryukhin, Effects of γ Radiation on Physio–Biochemical Modifications in Tubers and Plants, *Biokhim. Plodov i Ovoshchei, Akad. Nauk SSSR, Inst. Biokhim.*, **2**: 191–201 (1964). (In Russian)
35. M. Jaarma, Influence of Ionizing Radiation on Potato Tubers, *Ark. Kem.*, **13**: 97–105 (1958).
36. C. E. O'Hara, The Effects of Gamma Irradiation on the Dehydrogenase Activities and on the Proteins of Irish Grown Wheats, *Radiat. Bot.*, **9**: 33–38 (1969).
37. P. J. Bottino, A. H. Sparrow, and B. Floyd, manuscript in preparation.
38. A. H. Sparrow and E. Christensen, Improved Storage Quality of Potato Tubers After Exposure to Co^{60} Gammas, *Nucleonics*, **12**(8): 16–17 (1954).
39. A. H. Sparrow and L. Puglielli, Effects of Simulated Radioactive Fallout Decay on Growth and Yield of Cabbage, Maize, Peas and Radish, *Radiat. Bot.*, **9**: 77–92 (1969).
40. D. A. Bankes and A. H. Sparrow, Effect of Acute Gamma Irradiation on the Incidence of Tumor-Like Structures and Adventitious Roots in Lettuce Plants, *Radiat. Bot.*, **9**: 21–26 (1969).

41. K. A. Madsen, D. K. Salunkhe, and M. Simon, Certain Morphological and Biochemical Changes in Gamma-Irradiated Carrots (*Daucus carota* L.) and Potatoes (*Solanum tuberosum* L.), *Radiat. Res.*, **10**: 48–62 (1959).
42. J. P. Skou, Notes on Hyperplasia Induced in Carrots by β-Rays, Decapitation or *Agrobacterium tumefaciens*, *Radiat. Bot.*, **9**: 397–406 (1969).
43. N. Nybom, The Use of Induced Mutations for the Improvement of Vegetatively Propagated Plants, in *Mutation and Plant Breeding*, pp. 252–294, National Academy of Sciences—National Research Council, Publication 891, Washington, D. C., 1961.
44. J. Schubert, Mutagenicity and Cytotoxicity of Irradiated Foods and Food Components, *Bull. World Health Organ.*, **41**: 873–904 (1969).
45. P. J. Bottino and A. H. Sparrow, Effects of Exposure Time and Rate on the Survival and Yield of Lettuce, Barley, and Wheat, this volume.
46. M. J. Constantin, D. D. Killion, and E. G. Siemer, Exposure-Rate Effects on Soybean Plant Responses to Gamma Irradiation, this volume.
47. G. J. McCrory and P. Grun, Relationship Between Radiation Dose Rate and Lethality of Diploid Clones of *Solanum*, *Radiat. Bot.*, **9**: 27–32 (1969).
48. J. M. Bostrack and A. H. Sparrow, The Radiosensitivity of Gymnosperms. II. On the Nature of Radiation Injury and Cause of Death of *Pinus rigida* and *P. strobus* After Chronic Gamma Irradiation, *Radiat. Bot.*, **10**: 131–143 (1970).
49. E. J. Hall and J. S. Bedford, A Comparison of the Effects of Acute and Protracted Gamma-Radiation on the Growth of Seedlings of *Vicia faba*. I. Experimental Observations, *Int. J. Radiat. Biol.*, **8**: 467–474 (1964).
50. J. S. Krebs and G. F. Leong, Effect of Exposure Rate on the Gastrointestinal LD_{50} of Mice Exposed to ^{60}Co Gamma Rays or 250 kVp X-Rays, *Radiat. Res.*, **42**: 601–613 (1970).
51. A. H. Sparrow, B. Floyd, and P. J. Bottino, Effects of Simulated Radioactive Fallout Buildup and Decay on Survival and Yield of Lettuce, Maize, Radish, Squash, and Tomato, *Radiat. Bot.*, **10**: 445–455 (1970).
52. P. J. Bottino and A. H. Sparrow, Sensitivity of Lima Bean (*Phaseolus limensis* Macf.) Yield to ^{60}Co Gamma Radiation Given at Various Reproductive Stages, *Crop Sci.*, **11**: 436–437 (1971).
53. C. R. Davies, Effects of Gamma Irradiation on Growth and Yield of Agricultural Crops. I. Spring Sown Wheat, *Radiat. Bot.*, **8**: 17–30 (1968).
54. C. R. Davies, Effects of Gamma Irradiation on Growth and Yield of Agricultural Crops. II. Spring Sown Barley and Other Cereals, *Radiat. Bot.*, **10**: 19–27 (1970).
55. M. Devreux and F. Saccardo, Effets des radiations gamma sur les stades successifs de développement du gamétophyte mâle de tabac, *Radiat. Bot.*, **8**: 365–374 (1968).
56. M. Devreux and G. T. Scarascia Mugnozza, Effects of Gamma Radiation on the Gametes, Zygote and Proembryo in *Nicotiana tabacum* L., *Radiat. Bot.*, **4**: 373–386 (1964).
57. T. Hermelin, Effects of Acute Gamma Irradiation in Barley at Different Ontogenetic Stages, *Hereditas*, **57**: 297–302 (1967).
58. T. Kawai and T. Inoshita, Effects of Gamma Ray Irradiation on Growing Rice Plants. I. Irradiations at Four Main Developmental Stages, *Radiat. Bot.*, **5**: 233–255 (1965).
59. L. W. Mericle and R. P. Mericle, Radiosensitivity of the Developing Plant Embryo, in Fundamental Aspects of Radiosensitivity, Report of a Symposium, June 5–7, 1961, Upton, N. Y., USAEC Report BNL-675 (Brookhaven Symposia in Biology, Number 14), pp. 262–286, Brookhaven National Laboratory.
60. L. M. Monti and M. Devreux, Action des rayons gamma sur les gamètes, le zygote et le proembryon d'un pois fourrager, *Caryologia*, **17**: 433–441 (1964).

61. E. G. Siemer, M. J. Constantin, and D. D. Killion, Effects of Acute Gamma Irradiation on Development and Yield of Parent Plants and Performance of Their Offspring, this volume.
62. A. Yamashita, Effects of Acute and Chronic Gamma-Ray Irradiations on Growing Barley Plants, *Gamma Field Symp.*, **6**: 71–95 (1967).
63. A. H. Sparrow and S. S. Schwemmer, The Effect of Postirradiation Temperature on Survival Times in Herbaceous Plants Exposed to Gamma Radiation, *Radiat. Res.*, **43**: 227 (1970).
64. L. Decourtye, Stérilité, gamétique partielle provoquée par une irradiation gamma sur la variété de pommier Golden Delicious, *Radiat. Bot.*, **10**: 1–5 (1970).
65. T. D. Rudolph, Effects of X-Irradiation of Seed on X_1 and X_2 Generations in *Pinus banksiana* Lambert, *Radiat. Bot.*, **7**: 303–312 (1967).
66. A. H. Sparrow, L. A. Schairer, R. C. Sparrow, and W. F. Campbell, The Radiosensitivity of Gymnosperms. I. The Effect of Dormancy on the Response of *Pinus strobus* Seedlings to Acute Gamma Irradiation, *Radiat. Bot.*, **3**: 169–173 (1963).
67. F. G. Taylor, Jr., Predicted Seasonal Radiosensitivity of Southeastern Tree Species, *Radiat. Bot.*, **6**: 307–311 (1966).
68. G. N. Brown and F. G. Taylor, Interaction of Radiation and Photoperiodism in *Xanthium pensylvánicum.* Effects of Radiation During the Light and Dark Periods of Photoinduction, *Radiat. Bot.*, **7**: 67–72 (1967).
69. G. M. Clark, F. Cheng, R. M. Roy, W. P. Sweaney, W. R. Bunting, and D. G. Baker, Effects of Thermal Stress and Simulated Fallout on Conifer Seeds, *Radiat. Bot.*, **7**: 167–175 (1967).
70. J. F. McCormick, Changes in a Herbaceous Plant Community During a Three-Year Period Following Exposure to Ionizing Radiation Gradients, in *Radioecology*, Proceedings of the Symposium held Sept. 10–15, 1961, V. Schultz and A. W. Klement, Jr. (Eds.), pp. 271–276, Reinhold Publishing Corporation, New York, 1963.
71. J. F. McCormick and R. E. McJunkin, Interactions of Gamma Radiation and Other Environmental Stresses upon Pine Seeds and Seedlings, *Health Phys.*, **11**: 1643–1652 (1965).
72. L. N. Miller, Changes in Radiosensitivity of Pine Seedlings Subjected to Water Stress During Chronic Gamma Irradiation, *Health Phys.*, **11**: 1653–1662 (1965).
73. R. B. Platt, Ionizing Radiation and Homeostasis of Ecosystems, in *Ecological Effects of Nuclear War*, G. M. Woodwell (Ed.), USAEC Report BNL-917, pp. 39–60, Brookhaven National Laboratory, August 1965.
74. T. S. Osborne and A. O. Lunden, The Cooperative Plant and Seed Irradiation Program of the University of Tennessee, *Int. J. Appl. Radiat. Isotop.*, **10**: 198–209 (1961).
75. R. A. Nilan, *The Cytology and Genetics of Barley, 1951–1962*, Washington State University Press, 1964.
76. A. Ehrenberg, L. Ehrenberg, and G. Löfroth, Radiation-Induced Paramagnetic Centers in Plant Seeds at Different Oxygen Concentrations, *Abh. Deut. Akad. Wiss. Berlin, Kl. Med.*, **1962**(Nr.1): 229–231 (1962).
77. R. L. Latterell and D. M. Steffensen, The Oxygen Effect and Its Relation to Changes in Radiosensitivity During Seed Germination, *Mutat. Res.*, **4**: 191–200 (1967).
78. R. A. Nilan, C. F. Konzak, R. R. Legault and J. R. Harle, The Oxygen Effect in Barley Seeds, in *Effects of Ionizing Radiations on Seeds*, Conference Proceedings, Karlsruhe, 1960, pp. 139–154, International Atomic Energy Agency, Vienna, 1961 (STI/PUB/13).
79. R. A. Nilan, C. F. Konzak, J. Wagner, and R. R. Legault, Effectiveness and Efficiency of Radiations for Inducing Genetic and Cytogenetic Changes, *Radiat. Bot., Suppl.*, **5**: 71–89 (1965).

80. A. H. Sparrow and E. Christensen, The Effects of X-Ray, Neutron and Chronic Gamma Irradiation on Growth and Yield of Potatoes, *Amer. J. Bot.,* **37**: 667 (1950).
81. A. Heiken, Spontaneous and X-Ray-Induced Somatic Aberrations in *Solanum Tuberosum* L., *Acta Acad. Reg. Sci. Upsaliensis,* **7**: 1–125 (1960).
82. R. S. Kahan, Differential Effects of Small Doses of Ionizing Radiation on the Growth of Onion Seed and Bulblets and on Crop Yields, *Radiat. Bot.,* **9**: 175–178 (1969).
83. R. K. Schulz, Survival and Yield of Crop Plants Following Beta Irradiation, this volume.
84. B. Donini, G. T. Scarascia Mugnozza, and F. D'Amato, Effects of Chronic Gamma Irradiation in *durum* and Bread Wheats, *Radiat. Bot.,* **4**: 387–393 (1964).
85. M. Stoilov, G. Jansson, G. Eriksson, and L. Ehrenberg, Genetical and Physiological Causes of the Variation of Radiosensitivity in Barley and Maize, *Radiat. Bot.,* **6**: 457–467 (1966).
86. Y. Ukai, Studies on Varietal Differences in Radiosensitivity in Rice. III. Radiosensitivities with Respect to the Reduction in the Number of Dividing Cells and the Occurrence of Chromosome Bridges, *Jap. J. Breed.,* **18**: 221–228 (1968).
87. J. D. Spikes, Radiation Effects and Peaceful Uses of Atomic Energy in the Plant and Soil Sciences, in *Radioecology,* Proceedings of the Symposium held Sept. 10–15, 1961, V. Schultz and A. W. Klement, Jr. (Eds.), pp. 5–11, Reinhold Publishing Corporation, New York, 1963.
88. D. I. T. Walker and N. S. Sisodia, Induction of a Nonflowering Mutant in Sugarcane (*Saccharum* sp.), *Crop Sci.,* **9**: 551–552 (1969).
89. R. M. Alexakhin, R. T. Karaban, N. V. Kulikov, A. A. Molchanov, M. A. Naryschkin, S. V. Tarchevskaya, F. A. Tikhomirov, E. B. Tyuryukanova, and P. I. Yushkov, Some Aspects of Radioactive Fission Products Migration in the Forest Biogeocenoses and the Effects of Ionizing Radiations on the Woody Plants, in *Symposium International de Radioecologie,* Sept. 8–12, 1969, Cadarche, Report CONF-690918, Vol. 2, pp. 999–1023, Commissariat a l'Energie Atomique, 1970.
90. H. H. Smith, D. C. Joshua, N. C. Combatti, and K. H. Thompson, Relative Effects of Fission Neutron vs. X Irradiation of Seeds over a Wide Range of Genotypes, Doses and Moisture Levels, in *Symposium on Neutrons in Radiobiology,* USAEC Report CONF-691106, pp. 173–191, UT–AEC Agricultural Research Laboratory.
91. N. K. Notani and B. K. Gaur, Paradoxical Modifications in Radiosensitivity of Maize and Barley Seeds Stabilized for Different Moisture Contents, in *Biological Effects of Ionizing Radiation at the Molecular Level,* Symposium Proceedings, Brno, 1962, pp. 443–453, International Atomic Energy Agency, Vienna, 1962 (STI/PUB/60).
92. L. M. Josephson, data cited by T. S. Osborne and A. O. Lunden, The Cooperative Plant and Seed Irradiation Program of the University of Tennessee, *Int. J. Appl. Radiat. Isotop.,* **10**: 198–209 (1961).
93. C. R. Davies and S. F. Young, Effects of Irradiation of Parent Plants on Crop Yields, in Annual Report of Agricultural Research Council, Letcombe Laboratory, British Report ARCRL-20, pp. 12–15, 1969.
94. C. Y. Harn, J. K. Choi, and Y. S. Koh, Studies on Garlic Breeding Using Radiation. II. Morphological Effects on Gamma-Ray-Induced R_1 Generation in Garlic, *Allium sativum, J. Nucl. Sci. (Seoul),* **8**: 121–125 (1968). (In Korean)
95. H. Irizarry and J. Vélez Fortuño, Effect of Gamma Radiation Dosages on Rooting of Pineapple Crown Sections, Shoot Emergence and Growth, *J. Agr. Univ. Puerto Rico,* **54**: 551–558 (1970).
96. S. Matsumura and T. Fujii, Radiosensitivity in Plants. II. Irradiation Experiments with Vegetatively Propagated Plants, *Seiken Zihô,* **10**: 22–32 (1959).
97. W. S. Boyle, Radioresistance of F_1 Hybrids in the Triticeae, *Radiat. Bot.,* **8**: 1–6 (1968).

98. American Joint Committee on Horticultural Nomenclature, *Standardized Plant Names,* 2nd ed., J. Horace McFarland Co., Harrisburg, Penn., 1942.
99. D. D. Killion and M. J. Constantin, Fallout Radiation and Crop Productivity. Part B, UT–AEC Agricultural Research Laboratory Annual Report, OCD Work Unit 3223B, 1969.
100. Report of Committee 2 on Vulnerability of Crops to Fallout Beta Irradiation, Appendix A, this volume.
101. Report of Committee 3 on Vulnerability of Crops to Fallout Gamma Irradiation, Appendix A, this volume.